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Corrected, Updated, Lighter

PLAB 1 Keys is for PLAB-1 and UKMLA-AKT (Based on the New MLA Content-Map)

With the Most Recent Recalls and the UK Guidelines

ATTENTION: This file will be updated online on our website frequently!

(example: Version 2.2 is more recent than Version 2.1, and so on)

Key 1 **Extremely Important Collection for Neurology – PLAB 1**

These are just quick points to compare. Explanations are in the coming keys.

Cranial Nerves Nucleus:

 \vee 1, 2 \rightarrow Cerebral Cortex

 \vee 3, 4 \rightarrow Midbrain

v 5, 6, 7, 8 → Pons

 \vee 9, 10, 11, 12 \rightarrow Medulla

- Poorly controlled DM (nerve disorder) + Severe thigh or leg pain → followed by Proximal Muscular Wasting e.g. shoulders, thighs (quadriceps) muscles → Diabetic Amyotrophy.
- Chronic alcoholic + CAS (Confusion/ Ataxia/ Squint "Nystagmus, Ophthalmoplegia)
- → Wernicke's encephalopathy (Vitamin B1 -Thiamine- deficiency).
- → Give IV thiamine (vitamin B1).
- © Chronic alcoholic ± CAS + Amnesia (memory loss) + Confabulation (Making up stories) → Korsakoff's psychosis
- Important: brain MRI would show → Mamillary body atrophy. (Mamillary bodies and thalamic regions are responsible for amnesia -memory impairment-in patients who are chronic alcoholics who suffer from memory impairment Wernick's Korsakoff).
- OLD man + GDU (Gait abnormality/ Dementia -behaviour changes-/ Urine urgency ± incontinence) = Wet, Wobbly, Wacky Grandpa. (If CT/MRI is done, it would show enlarged ventricles mostly <u>WITHOUT</u> cortical atrophy).
- → NPH (Normal Pressure Hydrocephalus).

Next step → Perform lumbar puncture and CSF pressure monitoring.

Then \rightarrow CSF Shunt.

- OLD man + GDU (Gait abnormality/ Dementia -behaviour changes-/ Urine urgency ± incontinence) = Wet, Wobbly, Wacky Grandpa. But, with Hx of HTN, Smoking, TIAs, MRI shows multiple lacunar old infarcts
- → (Vascular Dementia).
- OLD + Making sexual or inappropriate comments/ Urinating on Sofa (*Disinhibition*), Loss of social interest (*disengagement*), Acting inappropriately or impulsively, Personality and behaviour changes, Over-eating, Struggling with word choices
- → Frontotemporal Dementia (Pick's disease)

The affected anatomical structure \rightarrow orbitofrontal lobe.

- Forgetful elderly (forget to lock doors, forget birthdays, forget names of people and places), easily getting lost (Disorientation), unable to do simple tasks (eg, cooking). (If CT/MRI is done, it would show enlarged ventricles mostly WITH cortical atrophy).
- → Alzheimer's disease
- **ACUTE** onset (hours to days) of mood and behavioural changes + Hallucinations (mainly visual)



- Elderly, UTI, or resp. infections, developed confusion, fluctuating level of consciousness and disoriented to time and place/ delusions/ hallucinations.
- → Delirium
- Old age + Bradykinesia (slow movements) + Resting tremors + Rigidity + Postural instability (may be falling forward) ± Expressionless face.
- → Parkinson's disease
- Parkinsonism (Parkinson disease features: Bradykinesia "slow movements"
- + Resting tremors + Rigidity + Postural instability "Ataxia", leaning forward)
- → Parkinson's Disease.
- Parkinsonism [+] Urinary incontinence, Erectile Dysfunction [±] Postural Hypotension "Frequent falls"
- → Shy-Drager Syndrome.

Mnemonic: "He is **Shy** as he wets his pants, has erectile dysfunction and **Drags** his feet because of his ataxia".

- Parkinsonism [+] Dementia [+] Visual Hallucinations ± Delusions
- → Lewy Body Dementia.

Investigations → **MRI brain** "to R/O other causes"

Followed by → **SPECT** (Single-photon emission computes tomography)

That is also known as (DatSCAN) ie, Dopamine transporter uptake imaging.

- Stiff- freezed- posture, Axial rigidity → Falling Backwards, shuffling and freezing gait, restricted downward gaze + Others
- → Progressive Supranuclear Palsy
- Tremors that are **ABSENT at rest and during sleep**, and **DO NOT resolve** with distraction + Worse when patient is tired or stressed. Start symmetrical, improve with alcohol consumption.
- → Essential Tremors (give Propranolol)
- Tremors that are absent at rest but resolve with distraction
- → Psychogenic tremors.
- AtaxiaIntentional Tremors (e.g. when trying to touch his nose or catch something)DysarthriaNystagmus. The affected part of the brain is
- → Cerebellum.

If limb ataxia → Cerebellar Lobe is affected.

If tRuncal ataxia \rightarrow Cerebellar VeRmis is affected.

■ Hx of GIT/ Respiratory infection (not always) +

Ascending Muscle weakness +

ABSENT or Diminished Reflexes

- → **Guillain Barre Syndrome**. (Autoimmune Demyelination)
- V The mechanism of Guillain barre syndrome
- → Autoimmune degeneration of myelin sheets of the peripheral neurons.

To $Dx \rightarrow Nerve conduction study$.

- Muscle Fatigue, Fatigue + Muscle Weakness + Normal reflexes
- ± D features (Drooping eyelids "ptosis", Diplopia, Dysphonia, Dys
- → To confirm Dx
- → Serum skeletal muscle nicotine acetylcholine receptor antibody
- If a presentation similar to Myasthenia Gravis but with (↑) reflexes (+ve for upper neuron signs) + normal autoimmune panel
- → Amyotrophic lateral Sclerosis. (ALS)
- Motor, sensory and reflexes are all affected but with typical LOSS OF PAIN AND TEMPERATURE (due to affection of Spinothalamic Tract) (e.g. one may burn his fingers without realising as he lacks Pain and Temp. sensation).
- → Syringomyelia
- **■** the same as Syringomyelia but [+] **CN involvement** (e.g. **Facial Palsy**).

- → Syringobulbia.
- Painless muscle weakness that worsens with exercise, **Tires easily**, Speech fades, Difficulty climbing stairs and reaching for items on shelves, Difficulty chewing and swallowing, Diplopia, **Normal reflexes** ± FHx of an **autoimmune** disease (eg, Thyroid disease) (**Positive autoimmune panel**)
- → Myasthenia Gravis
- → To confirm Dx
- → Serum skeletal muscle nicotine acetylcholine receptor antibody
- **OPTIC NEURITIS** (pale disc, swollen disc, blurred vision...) + Muscle **Stiffness** + Muscle **Weakness** + Others ± Hx of attacks.
- → Multiple Sclerosis.
- Wasted tongue with fasciculations + Bovine cough (unable to produce expulsive cough due to inability to close epiglottis), reduced gag reflex
- → Bulbar Palsy.
- Multiple Cranial nerve affection (e.g. Facial numbness "5th CN Trigeminal nerve" + Diplopia and Ptosis "3rd CN Oculomotor nerve")
- → Brainstem lesion

Oculomotor (3rd CN) \rightarrow Midbrain Trigeminal (5th CN) \rightarrow Pons

- Symptoms of (Cerebellar Lesion) e.g. ataxia -unsteadiness-, nystagmus, slurred speech
- [+] Cranial Nerves Symptoms (e.g. Vertigo: 8th CN Vestibulocochlear nerve Diplopia: 3rd CN Oculomotor nerve)
- → Brain Stem lesion
- Ipsilateral oculomotor nerve palsy (e.g. left eye ptosis, left mydriasis -dilated pupil-, left eye is deviated outwards and downwards)
- [+] Contralateral hemiparesis (e.g. right arm + right Leg weakness)
- → Weber's Syndrome (MIDBRAIN infarct) (PCA block: Posterior Cerebral Artery)
- **Ipsilateral** Horner's syndrome + Loss of Pain and Temperature sensation in Face
- [+] Contralateral loss of Pain and Temperature sensation in Limbs.
- → Wallenberg's Syndrome = Lateral MEDULLARY Syndrome (PICA Block: Posterior Inferior Cerebellar Artery).
- Sudden-onset of **nystagmus**, **gait ataxia**, **vertigo Dizziness**-, homonymous **hemianopia**, **dysdiadochokinesia**:
- √ The likely $Dx \rightarrow Posterior Circulation Stroke.$
- √ The Ix of choice → MRI of the head [not CT].
- Generally, in acute stroke → CT without contrast (but it is of a <u>limited value</u> in posterior circulation stroke).
- Other features of Posterior Circulation Stroke

→ Nausea, vomiting, dysarthria, gaze-evoked jerk nystagmus, unilateral limb weakness and numbness.

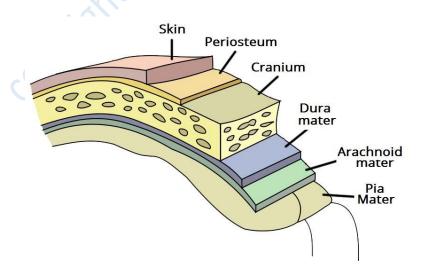
Dysdiadochokinesia = inability to perform rapid alternating muscle movements.

- Contralateral Paralysis and Sensory Loss (Face and Arm)
- + Ipsilateral Gaze Preference
- → Middle Cerebral Artery (MCA) Stroke

More Elaboration and Explanation on each of these points are in the coming keys.

Key

Headache due to Brain Injury



Epidural "Extradural" Hematoma"

- ♦ Often associated with Skull Fracture.
- **♦ Middle Meningeal artery** injury.
- **♦ Lucid interval**:

A patient goes back to do what he was doing before falling unconscious again.

Chronic Subdural hematoma

- ♦ Usually an elderly ± on Anticoagulant (eg, warfarin) or Alcoholic.
- **♦** Hx of minor fall ± Minor head injury.
- ◆ **SLOW** onset of symptoms.
- ♦ CT is diagnostic, and Surgical evacuation of hematoma provides dramatic improvements.
- ◆ Note: Acute Subdural hematoma resembles Epidural hematoma (lucid interval).
- ♦ Important → The affected vessel → Bridging Vein (Cerebral Vein).

Subarachnoid hemorrhage

- ♦ Occurs most commonly due to cerebral ANEURYSM.
- **♦** Common associations:
- √ Ehlers-Danlos Syndrome (collagen problem "Connective Tissue Disease").
- √ Polycystic Kidney Disease (ADPKD) (Hypertension and repeated kidney stones) → Association: Berry Aneurysm,

Association \rightarrow SIADH \rightarrow Hyponatremia. (Important \checkmark)

V Excessive alcohol intake is an important risk factor. ■

- ♦ Usually **Sudden** and **Spontaneous**, **Very Severe** ± **may be associated with nausea**, **vomiting**, **photophobia**.
- **♦** Common hints:
- **V** The worst headache in life.
- **V** Thunderclap headache.
- **V** Headache of severe intensity/ sudden onset.
- √ Feeling as "kicked in the head" (SEVERE headache worse at back of head).
- √ Severe "Occipital".
- √ Meningeal irritation (Neck stiffness, Photophobia), Vomiting, Collapse, Seizures. Fever might also occur due to blood irritation to the meninges.
- Diagnosing Subarachnoid Haemorrhage (SAH):
- **∨** CT scan of the brain (without contrast)
- √ if inconclusive → LP "Lumbar Puncture" (CSF is Bloody, then → Xanthochromic "Yellow" due to bilirubin). "Important √"

Important: CT scan is NOT ALWAYS conclusive of SAH. Thus, if the CT is negative but the history still goes with SAH, the go for \rightarrow Lumbar puncture.

Note, **calcium channel blockers** eg, **Nimodipine**: (for 5-14 days): useful to diminish the anticipated cerebral vasospasm that may occur with SAH.

Elaboration

Example (1),

A 40 YO \bigcirc presents with a sudden onset of a severe headache and recurrent episodes of vomiting. She suddenly collapsed one-hour after taking paracetamol. Her medical Hx is significant for Ehlers Danlos Syndrome.

The likely Dx → Subarachnoid Hemorrhage

Sudden onset, Severe, Association with Ehlers Danlos Syndrome

Example (2),

A 35 YO man was hit by a car and lost consciousness. When the paramedics arrived, he was found to regain consciousness. However, his GCS suddenly deteriorated again and went into unconsciousness while in the A&E with his left pupil being dilated and unresponsive to light.

The likely $Dx \rightarrow Epidural$ (Extradural) Hemorrhage.

√ The patient went into lucid interval then he went to unconsciousness again.

 ✓ Mass effect due to hematoma (space-occupying lesion: hematoma, abscess, tumor) → Unilateral dilated unreactive pupil.

A Quick Reminder:

- Unilaterally Dilated pupil → Space-occupying lesion (eg, abscess, tumour, hematoma).
- Bilaterally Constricted pupils (bilateral pinpoint = Miosis)
- → Opiates overdose (eg, morphine, Heroin) CVA affecting the brainstem.
- **Bilaterally Dilated pupils** → Amitriptyline (TCA) overdose Cocaine overdose.

Example (3),

While playing rickets, a 24 YO girl was hit in the head by the ball. She initially lost consciousness but spontaneously recovered. Now, she has severe headache with an episode of vomiting and slow responses.

The likely Dx → Epidural (Extradural) Hemorrhage

Again, **lucid interval** is the key.

Example (4),

68 YO man becomes confused over a period of 2 weeks. He used to be active and go for long walks. Now, he stares at the wall, barely talks to anyone, and sleeps the majority of the day. His daughter recalls that he fell down the stairs about a week before these changed happen.

The likely diagnosis → Chronic Subdural Hemorrhage

• Chronic progressive symptoms, Elderly, Hx of fall (see above).

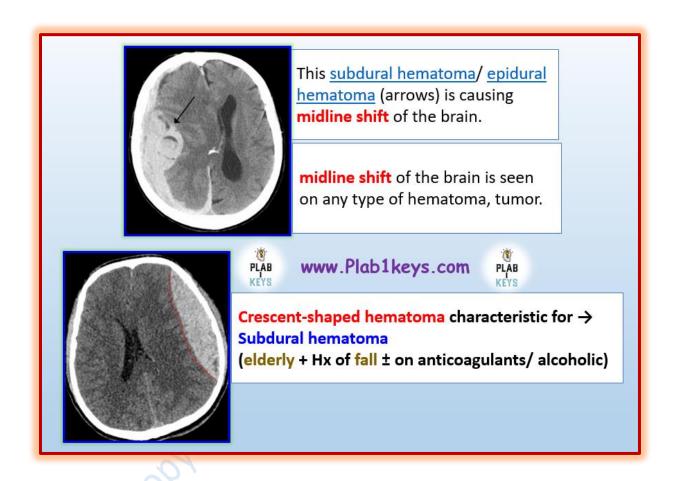
Example (5),

A lady found her 68 YO father at home in a confusional state with bruising on his left arm and unsteady gait. CT scan brain show a midline shift from the side of a clot.

The likely Dx → Subdural Hemorrhage

Elderly + Recent fall (bruises) → think of subdural hematoma.

Note, **all 3 types** of hematoma can present with (**midline shift**); however, the presence of "<u>Crescent-Shaped hematoma</u>" is characteristic for **subdural hematoma** (not mentioned here).



Example (6),

65 YO \bigcirc Presents with Severe headache with nausea and vomiting for the last 12-hours. She describes it as (Kicked in the head) that is worse at the back of the head. CT scan of head shows no abnormalities.

The best next step \rightarrow Lumbar Puncture (LP)

Here, we suspect SAH "Subarachnoid hemorrhage" Clinically.

First line \rightarrow Non-contrast CT, if it is inconclusive \rightarrow LP, still? \rightarrow MRI

After the diagnosis of SAH has been confirmed, we need to identify the <u>origin</u> (site) by either \rightarrow Cerebral Angiography (or) CT Angiography.

Example (7),

A 56 YO chronic alcoholic has been found confused by his wife. He had fallen a week ago. Since then, he has worsening agitation and confusion.

The likely $Dx \rightarrow$ Subdural hematoma.

Chronic Alcoholic + Hx of fall + Slow onset/ progress of symptoms

Example (8),

A 70 YO \bigcirc with a Hx of fall 3 days ago presents with progressive confusion, drowsiness and disorientation.

- ◆ The likely Dx → Chronic Subdural Hemorrhage.
- ♦ The likely involved blood vessel
- → Cerebral Vein (Rupture of a bridging vein).

Example (9),

A daughter has brought her 77 YO mother to a clinic complaining that her mother is progressively having worsening confusion lately. The old lady is on Warfarin for AF and her INR is 6.8. She has bruises on her arms.

The likely diagnosis → Chronic Subdural Hemorrhage.

- Chronic progressive symptoms, Elderly on Anticoagulant (warfarin), Hx of fall (Arm bruises). (see above).
- Any minor fall can cause bleeding in those who are on anticoagulation regimen, particularly those who have high INR.
- This lady will need to stop warfarin, to be given IV vitamin K1 + **Prothrombin complex concentrate**. This is because she has major bleeding.

Example (10),

A 67 YO \circlearrowleft fell down the stairs. His son brought him the ED. He was having lucid intervals and then he went unconscious.

The likely vessel affected → Middle meningeal artery

 \forall Lucid intervals \rightarrow Epidural hemorrhage \rightarrow Middle meningeal artery.

V Remember, in chronic subdural hemorrhage → Cerebral "Bridging" veins.

Example (11),

A 52 YO lady presents with sudden onset, severe occipital headache with vomiting and neck pain. CT is inconclusive. LP shows Xanthochromia.

The likely $Dx \rightarrow$ Subarachnoid hemorrhage.

∨ CT brain (without contrast)

V if inconclusive → LP "Lumber Puncture" (CSF is Bloody, then → Xanthochromic "Yellow" due to bilirubin). "Important V"

Example (12),

A 25 YO \circlearrowleft was found on the floor. He is drunk, drowsy and unsteady. On admission to the ED, he has a sudden onset of headache of severe intensity

that is localised to the occipital area. GSC is 15/15 and with no neurological deficits.

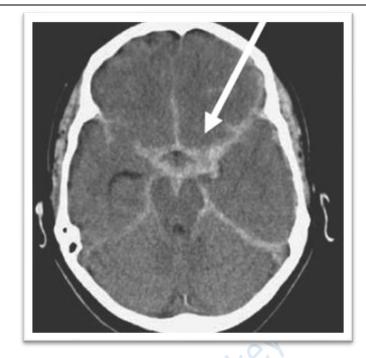
The likely $Dx \rightarrow$ Subarachnoid Hemorrhage (SAH)

Sudden, Severe, Occipital, Alcohol (all are hints towards SAH).

Review the topic above.

Example (13),

■ A 36 YO man presents to the ED with a Severe headache with vomiting for 1 day. The headache started when he was lifting weights in a gym. He has photophobia and neck stiffness and GCS of 12/15. A CT head is ordered and it shows:



His BP is normal with mild tachycardia. Which drug is useful in this case?

[Aspirin or: Clopidogrel or: Sumatriptan or: Nimodipine]

√ Firstly, this is a case of Subarachnoid hemorrhage (SAH).

 \lor The hyperintense areas on the CT \rightarrow blood in the subarachnoid basal cisterns.

V In SAH, cerebral vasospasm can occur 4-12 days later and it is serious.

- To diminish this anticipated cerebral vasospasm,
- → we give Calcium Antagonist (e.g. Nimodipine).

Example (14),

■ A 53 YO woman presents to the A&E complaining of severe headache, photophobia and nick stiffness. The headache started suddenly 3 hours ago and she took sumatriptan which did not relieve the headache. She reports that 1 week ago she had severe headache but was not as intense as this one. Also, she mentions that over the past week, she has had several pulsating headaches for which she took sumatriptan. Her body temperature is 38.6.

The likely $Dx \rightarrow$ Subarachnoid hemorrhage (SAH).

- \checkmark Severe/ More **intense** than ever \rightarrow points towards SAH.
- **V** Sudden onset headache \rightarrow points towards SAH.
- \checkmark SAH \rightarrow Meningeal irritation \rightarrow Nick stiffness, photophobia, fever.
- The fever here is likely due to blood irritating the meninges.
- The intermittent headaches during the previous week are likely due to sentinel bleeds (leakage of blood into subarachnoid space which may occur before the aneurysm rupture).
- ▼ Sudden onset/ intense favour SAH over meningitis which has gradual onset.
- Migraine does not have nick stiffness and fever.

Example (15),

■ A 46-year-old woman presents to the A&E complaining of severe headache associated with nausea, vomiting and photophobia that started 15 hours ago. There is no fever and no history of trauma. Her neurological examination is normal. She was given analgesics and undergone CT scan which revealed no acute abnormalities. What is the most appropriate action?

→ Lumbar puncture

Sudden and Spontaneous, Very Severe ± may be associated with nausea, vomiting, photophobia → Think: Subarachnoid haemorrhage.

Diagnosing Subarachnoid Haemorrhage (SAH):

∨ CT scan of the brain (without contrast)

v if inconclusive → **LP** "**Lumbar Puncture**" (CSF is **Bloody**, then → **Xanthochromic** "**Yellow**" due to bilirubin). "**Important v**"

Important: CT scan is NOT ALWAYS conclusive of SAH. Thus, if the CT is negative but the history still goes with SAH, the go for → Lumbar puncture.

Key

CSF findings in Meningitis

Bacterial	Viral	Tuberculous
Meningitis	Meningitis	Meningitis

Glucose	Low	Normal	Low
Protein	High	Normal/ High	High
WBCs	Mainly Neutrophils	Mainly Lymphocytes	 Early → Neutrophils. Later → Lymphocytes.

- ♦ In **Bacterial meningitis**, the bacteria eat glucose → Low glucose
- + rapidly destroy tissues \rightarrow High Protein. + (\uparrow Neutrophils).
- ♦ In **Viral Meningitis**, the virus does not eat glucose → Normal Glucose
- + slowly destroy tissues \rightarrow Normal or High protein. + (\uparrow Lymphocytes).
- ♦ Mycobacterium Tuberculosis is bacteria and thus the glucose is low. However, the difference that in ordinary bacterial meningitis, there is ↑ Neutrophils whereas in TB meningitis, there could be lymphocytosis (↑ Lymphocytes).

Important Notes about meningitis organisms:

- Gram +ve diplococci → Streptococcus pneumoniae.
- Gram -ve diplococci → Neisseria Meningitidis.
- Gram +ve cocci in grape-like clusters, Coagulase and Catalase positive

- → Staphylococcus aureus.
- Gram +ve bacilli → Listeria monocytogenes.
- **□** Gram -ve coccobacilli \rightarrow H. influenza.

Also,

 \forall Turbid (or purulent) CSF (+) No rash \rightarrow think streptococcus pneumoniae.

V Turbid (or purulent) CSF (+) there is rash → think Neisseria Meningitidis.

Elaboration

Example (1),

A 36 YO \circlearrowleft presents with gradually increasing headache, photophobia and neck stiffness. CT scan is normal. LP is done and the CSF shows low glucose with lymphocytosis. There is no organism on stain.

The likely causative organism \rightarrow Mycobacterium Tuberculosis.

√ (Low glucose + ↑ Lymphocytes) → TB meningitis.

- √ (Low glucose + ↑ Neutrophils) → Bacterial meningitis.
- √ (Normal glucose + ↑ Lymphocytes) → Viral meningitis.

Example (2),

25 YO ♀ presents with 1-day headache that has been increasing in severity. She cannot tolerate bright light and there is discomfort on turning her head. Kernig's sign is negative. Lumber puncture CSF shows: Normal glucose, elevated protein and lymphocytosis.

The likely diagnosis → Viral meningitis.

Note

 \checkmark Kernig's sign does not have to be positive in all cases of meningitis.

This patient has headache, photophobia, neck rigidity and LP findings suggesting meningitis.

√ (Normal glucose + ↑ Lymphocytes) → Viral meningitis.

Key 4

Commonly Asked Types of Headache

Tension VS Migraine VS Cluster

Tension Headache

- \square Location: \rightarrow Bilateral.
- Pain Characteristic: → Pressing/ Tightening/ Dull.
- \square Pain intensity: \rightarrow Mild to Moderate.
- Other associated Symptoms: None.
- \square Duration: \rightarrow Around 30 minutes.
- Patient appearance: → Patient is able to do daily activities.
- Management: → Pain killers (Aspirin, NSAIDs, Paracetamol).
- lacktriangledown Prophylaxis: \rightarrow Acupuncture.

Migraine Headache

- **□** Location: → Unilateral (70%) **■** Bilateral (30%).
- Pain Characteristic: → Throbbing/ Pulsating Gradual onset.
- Pain intensity: → Moderate to Severe.
- Other associated Symptoms: Photophobia, Nausea/Vomiting,

Aura/ Triggers: (Visual symptoms, Sensory symptoms, Speech disturbance).

- \square Duration: \rightarrow 4-72 hours.
- Patient appearance:
- → Prefers to sit in a dark, quiet room. Limited activities.
- Management (Acute phase):
- \forall First line \rightarrow Sumatriptan (If > 17 YO \rightarrow Oral). (12-17 YO \rightarrow Nasal).

If the patient is **vomiting** (cannot tolerate orally) \rightarrow **SC** sumatriptan.

- \forall Second line \rightarrow NSAIDs (eg, Aspirin).
- √ Combination therapy:

(Oral sumatriptan + NSAIDs [or] Oral sumatriptan + Paracetamol).

√ Transcutaneous vagus nerve stimulation.

√ Avoid triggers.

Prophylaxis: →**Beta-Blockers** (eg, **Propranolol**) [or] **Topiramate**.

Important note:

If a question asks about the **first line medication** to reduce the frequency of the **symptoms**, do not rush it and pick sumatriptan! The question means what is the first line medication used as <u>prophylaxis</u> (to reduce the frequency of attacks) which is **beta blockers** (eg, **propranolol**).

Sumatriptan is used in "acute" attack of migraine.

Important note:

The <u>disappearance</u> of Migraine for a long period can occur and a <u>recurrence</u> can occur long time later.

In this case \rightarrow No Further Investigations are required. It is migraine again.

However, if, after disappearance, a <u>different</u> headache (**e.g**. with neck stiffness, or described as the worst headache in life, we should request CT scan head to R/O other causes of headache such as <u>Subarachnoid</u> hemorrhage).

Cluster Headache

```
■ Location: → Always Unilateral.
Pain Characteristic:
→ Variable Pain on one eye Sudden onset and recovery.
■ Pain intensity: → Very Severe (Excruciating)
Other associated Symptoms
Ipsilateral watery/ red eye constricted pupil rhinorrhea facial
sweating drooping eyelid (ptosis). (Photophobia, vomiting can also occur).
■ Duration: → 15 minutes to 3 hours
From one attack each 2 days up to 8 attacks a day!
Recurrent attacks affect the same usual side.
\blacksquare Patient appearance: \rightarrow Restless patient with inability to do activities.
Management (Acute phase):
√ 100% O₂ for 10-20 minutes.
√ Sumatriptan (Nasal or Subcutaneous).
```

V If first time attack → refer to specialist as it may require CT to R/O other differential diagnoses.

Prophylaxis (Chronic): → Calcium Channel Blockers (eg, Verapamil).

Elaboration with Scenarios

Example (1),

34 YO \bigcirc presents with a Hx of recurrent attacks of intense headaches. They are usually unilateral, pulsating, and associated with photophobia and vomiting.

 \forall The likely $Dx \rightarrow Migraine$.

V The first line treatment → ORAL Sumatriptan.

Sumatriptan in Migraine \rightarrow Oral.

Sumatriptan is Cluster headache → Nasal/ Subcutaneous.

Example (2),

A 42 YO \circ presents with a severe right-sided throbbing headache and photophobia 20 minutes after an episode of tingling and numbness of left hand.

→ Migraine with Aura (Classical).

Example (3),

A 19 YO \bigcirc has episodic headaches preceded by fortification spectra and can sometimes last for 2-3 days.

The most appropriate treatment in the acute phase

→ Oral Triptans.

What if triptan was not given in the options?

→ Aspirin (or NSAIDs).

- This is a case of Migraine.
- Fortification spectra are more complicated images that can float in your vision that can proceed a migraine (Aura).
- Remember, **Topiramate** and **Propranolol** (Beta-Blockers) are not used in acute phase; they are **preventative** (**prophylactic**) medications in migraine.
- Do not pick Topiramate thinking it is triptan. They are different!

Example (4),

30 YO man presents complaining of severe headache in the last 2 hours ago. It is localised to the left side of his head and associated with photophobia. He suffered from a similar episode a few months ago that had also involved the left side. It had lasted for several minutes for several times a day for a period of 2 weeks and then subsided spontaneously. The patient is unable to stand still, agitated and anxious.

The likely $Dx \rightarrow \underline{\text{Cluster headache}}$.

- Do not get fooled by "Photophobia". It can also occur in cluster headache.
- Remember, for cluster headache:

√ Duration: → 15 minutes to 3 hours

From one attack each 2 days up to 8 attacks a day!

Recurrent attacks affect the same usual side.

√ Patient appearance:

→ Restless agitated patient with inability to do activities.

These are compatible with this scenario:

- The previous attacks lasted for **several minutes** and occurred **SEVERAL times a day** (which is seen more commonly in cluster headache).
- Think of cluster headache as a visitor who visits frequently for a month or so and then disappear. That's why it is called (Cluster). Many frequent back-to-back attacks during a limited period, then bam, disappear, and re-visit again after a while.
- Remember in migraine, the duration is Longer (4 hours to 72 hours)!

√ The patient is **agitated** (while in migraine, the patient usually prefers to lay down in a dark room).

Example (5),

While he is working in his office, a 31 YO \circlearrowleft suddenly developed excruciating headache to his left side associated with left eye pain. He experienced similar episodes 3 months ago. His left eye is red, swollen and with lacrimation.

The likely $Dx \rightarrow$ Cluster headache.

Excruciating = very sever, eye pain swelling redness and with lacrimation are features of cluster headache. The Hx of similar attacks is also important. Always same side.

Example (6),

A 30 YO female presents complaining of recurrent headaches. These headaches usually unilateral, last for 24-48 hours, have pulsatile character, associated with nausea and photophobia, and usually requires her to limit her activities.

The likely $Dx \rightarrow Migraine headache$.

The 1st line $Rx \rightarrow \overline{\textbf{ORAL sumatriptan}}$.

✓ Pulsatile, lasting longer than 4 hrs, associated with nausea and photophobia
 → Migraine.

V Cluster headache lasts from 15 minutes to 3 hrs.

Remember

V in migraine: > 17 YO \rightarrow oral triptans
■ < 17 YO \rightarrow nasal triptans.

√ in **cluster** headache: nasal or SC triptans.

Example (7),

A 27 YO female presents complaining of severe unilateral headache associated with nausea, vomiting and light sensitivity. These attacks occur every couple of weeks and last for several hours. What is the first-line medication that can help reduce the frequency of these attacks?

- **√** The likely Dx here is **Migraine**.
- ✓ Note that the question asks about a treatment used as **prophylaxis** (to reduce the frequency of the symptoms). In this case:
- **V** The first-line prophylactic medication is → Beta blockers (Propranolol).
- ✓ If the question asks about the first-line management in **acute attack**, the answer would be (oral sumatriptan: if >17 YO) or (Nasal sumatriptan: if 12-17 YO) with or without NSAIDs/paracetamol.

Example (8),

A 36 YO woman presents to a neurology clinic complaining of episodes of temporary weakness of left arm and numbness on her left face. This is followed by headache on left side of her head. The episodes are recurrent around twice a month, each can last for up to 24 hours. Her blood pressure is normal. What is the most suitable PROPHYLACTIC medication?

- **√** The likely Dx here is **Hemiplegic migraine with aura**.
- ✓ Note that the question asks about a treatment used as **prophylaxis** (to reduce the frequency of the symptoms). In this case:
- **V** The first-line prophylactic medication is → Beta blockers (Propranolol).
- ✓ If the question asks about the first-line management in **acute attack**, the answer would be (oral sumatriptan: if >17 YO) or (Nasal sumatriptan: if 12-17 YO) with or without NSAIDs/paracetamol.

Key 5

Multiple Sclerosis (MS)

- Multiple sclerosis is **chronic** cell-mediated **autoimmune** disorder characterised by **demyelination** in the **central** nervous system.
- 3 times more common in women
- Most commonly diagnosed in people aged 20-40 years
- \blacksquare The Commonest Type \rightarrow Relapsing-remitting MS (> 85%):

Acute attacks (e.g. last 1-2 months) followed by periods of remission.

Important Features: Important Features:

Visual

Optic neuritis: common presenting feature (usually given as a hint in exam √)

Sensory

- Pins/needles
- Numbness
- Trigeminal neuralgia
- Paraesthesia in limbs on neck flexion

Motor

Spastic weakness: most commonly seen in the legs (important √)

Cerebellar → Ataxia | Tremors | Dysarthria | Nystagmus

Others

Urinary urgency/ retention

Sexual dysfunction

Intellectual deterioration

Another way to memorise them:

Motor and Sensory symptoms due to affection of (Cerebellum + Brainstem + Transverse Myelitis)

♦ Cerebellum → Ataxia | Tremors | Dysarthria | Nystagmus.

If limb ataxia → Cerebellar Lobe is affected.

If Truncal ataxia → Cerebellar VeRmis is affected "Midline of cerebellum"

- ◆ **Transverse Myelitis** → Weakness (e.g. in legs), Spastic Quadriparesis or Spastic Paraparesis Urinary urgency/ retention Stiffness ↑ tone and brisk reflexes.
- Most important hint → Optic neuritis / Pale disc / Acute unilateral visual reduction that might be painful.
- √ Note, the white matter of the optic nerve, brainstem and basal ganglia are most commonly affected in Multiple Sclerosis.

Paresis = Weakness/ Partial paralysis. (Paraparesis: Lower limbs). (Quadriparesis: all 4 limbs)

Diagnosis:

√ Clinical diagnosis.

 \lor Definitive Diagnostic test \rightarrow MRI \rightarrow Demyelination, Lesions disseminated in time and place.

Treatment:

√ In acute cases (during a Relapse) (initial) → Oral or IV Methylprednisolone.

V Long-term → Glatiramer acetate [or] Interferon-beta.

Example (1),

25 YO \bigcirc presents complaining that she has a progressive stiffness of her left leg. She also mentions that she has recurrent blurry vision that resolves spontaneously each time. Ophthalmoscopy reveals pale disc. LL examination reveals 3/5 left leg power with upward planters.

The best initial treatment \rightarrow **Methylprednisolone**.

The features in this stem:

Stiffness (upward planters) + Weakness (low power) in LL + Optic Neuritis (Blurry vision + Pale disc) that is remitting and relapsing → Multiple Sclerosis.

So,

Stiffness + Weakness + Optic Neuritis + Recurrence/ intermittent → MS

Example (2),

A 44 YO \bigcirc presented with blurred vision and intermittent clumsiness for 3 months. Reflexes are brisk in her arm and the optic disc is pale.

The likely Dx → Multiple Sclerosis (Intermittent + Optic Neuritis)

The best modality to confirm $Dx \rightarrow MRI brain$

The best drug in acute phase → Methylprednisolone

Key 6

- Dizziness/Vertigo on Moving head 9for seconds to minutes) → BPPV.
- Add: Hx of Viral URTI → Vestibular Neuritis.
- Add: Hearing loss/Tinnitus → Labyrinthitis.

Another Summary:

- Vertigo + Hx of Common cold → Vestibular neuritis.
- Vertigo + Hx of Common cold + Hearing loss/Tinnitus → Labyrinthitis.
- Vertigo on moving head that lasts for seconds WITHOUT Hx of Common cold
 → Benign Paroxysmal Positional Vertigo (BPPV).
- DVT + Fullness (Deafness, Vertigo, Tinnitus) + Pressure or Fullness in one ear
 → Meniere's disease (MRI is normal).
- DVT + Cranial Nerve Palsy (e.g. Facial palsy, Loss of Corneal reflex, Loss of facial sensation) ± Fullness/ pressure feeling.
- → Acoustic Neuroma (Vestibular Schwannoma)

(Do MRI of the cerebellopontine angle).

Benign Paroxysmal Positional Vertigo

- Sudden onset of vertigo that is **aggravated by a change in head position** and lasts a **few seconds** to a few **minutes** ± Nausea.
- Occurs usually during turning over in bed or lying down.

- The sensation that the room is spinning.
- · $Dx \rightarrow Hallpike's Manoeuvre$.
- · Rx → Epley's Manoeuvre = repositioning technique. "Apply Epley for Treatment"
- N.B. Mostly resolves spontaneously.

Benign paroxysmal positioning vertigo (BPPV) is a peripheral vestibular disorder involving the **semicircular canal** usually but not exclusively the **posterior semi-circular canal** (PSCC). **V imp.**

Key Drop Attacks → Sudden falls WITHOUT losing consciousness due to either cauda equina (weak legs) or vertebrobasilar insufficiency. The whole event is remembered. The recovery is complete and fast.

Stokes Adam attack → Sudden collapse into "Unconsciousness" due to a disorder of heart rhythm in which there is a slow or absent pulse resulting in syncope (fainting) with or without convulsions.

DDx of Sudden Falls.

- **Drop attacks** → Sudden falls without losing consciousness.
- Stokes Adam → Unconscious + Abnormal ECG.

- **Hypoglycemia** → Unconscious (or) ↓ level of consciousness + Sweating/tachycardia, do not recover unless given glucose.
- Vasovagal attacks

 Unconscious + Hx of prolonged standing, straining, pooping, heavy weight lifting or after visual stimuli e.g. seeing blood. The patients usually feel dizzy and "tunnel vision" before the attack. Usually in a YOUNG FEMALE (with NO chest pain, palpitation and with Normal ECG)
- **Epilepsy** → Unconscious ± Post-seizure confusion

Key 8

Wernicke's encephalopathy (Thiamine deficiency)

- In Chronic Alcoholics mainly.
- Other causes: **Persistent vomiting** (hyperemesis gravidarum in pregnancy), Stomach Cancer.
- IMPORTANT → the cause: Vitamin B1 deficiency (Thiamine).
- Triad of CAS: Chronic Alcoholic/ Chronic severe vomiting +
- Confusion
- Ataxia (Uncoordinated gait, unsteadiness)
- Squint (Nystagmus, Ophlamoplegia)
- Rx → Urgent IV Thiamine (Vitamin B1) even before glucose replacement.
- If not treated → It might develop to Wernicke's Korsakoff Syndrome =

Korsakoff Psychosis

(The above CAS triad + Amnesia + Confabulation).

Confabulation = the patient makes up stories to replace the forgotten details (he is not lying; he thinks that these stories have truly occurred).

They may carry on a coherent conversation, but moments later, they cannot remember that they have a conversation.

NOTE: Sometimes **not all** features are given in a stem. When you see a **chronic alcohol** consumer with **Confusion** (loss of concentration) + Antero- or Retrograde **Amnesia** + **CONFABULATION** (Making up stories), think of → **Korsakoff Psychosis**.

Important Question (Previously Asked):

What is expected to be seen on the MRI of a chronic alcoholic who suffers from Amnesia (memory impairment)? In other words, Wernicke's Korsakoff syndrome can be seen in chronic alcohol consumers. One of its features is Amnesia (memory impairment). Which brain structures that are responsible for this amnesia?

→ Mamillary bodies and thalamic regions. (Responsible for memory).

(MRI would show → Mamillary bodies atrophy).

Key 9

Stroke

→ Sudden focal neurological loss due to vascular origin lasting for > 24 hrs.



→ Sudden focal neurological loss due to vascular origin lasting for < 24 hrs.

2ry Prevention (Long-term management) of Ischemic Stroke/ TIA:

- ✓ Control Blood Pressure.
- Statins (for All patients regardless of their cholesterol baseline level).
- Ani-platelets (or) Anti-coagulation:

(Based on presence or absence of AF):

- If there is Atrial Fibrillation \rightarrow Anticoagulants: Warfarin [or] DOAC (Dabigatran/ Apixaban/ Rivaroxaban/ Edoxaban).
- If **No Atrial Fibrillation** → Antiplatelets: **Clopidogrel** 75 mg OD.

<u>Important</u>, in <u>Acute</u> **presentation of stroke**, we initially need to perform [CT scan head] to exclude hemorrhage (to confirm that it is **ischemic** not hemorrhagic stroke).

If ischemic and presenting <4.5 hrs \rightarrow give Alteplase "Thrombolytics".

<u>Important</u>, after alteplase, we then give Aspirin 300 mg for the first 2 weeks, then we give clopidogrel 75 mg for life.

To sum up, in "ischemic stroke" where there is no AF:

Alteplase (if presenting <4.5 hrs of the onset of the symptoms). Then

- → Aspirin 300 mg (for 2 weeks). Then:
- \rightarrow Clopidogrel 75 mg (for life).
- Lifestyle advice \rightarrow low salt, fat, alcohol, quit smoking, weight loss.

Important Note:

What if the question mentions that the symptoms resolved in <24 hours?

- → This is a Transient ischemic attack -TIA- (not a stroke).
- → CT scan head to exclude hemorrhage.
- → Give Aspirin (for 2 weeks, then change it to clopidogrel 75 mg)

(In TIA, we do NOT give alteplase - ie, no thrombolysis in TIA - Be careful)!

→ TIA requires Carotid doppler scanning. (CT Angiography of carotid vessels).

Example (1),

A patient had slurred speech for 5 minutes. He has hypertension and he takes medications for it. His ECG is normal (sinus rhythm) with HR of 80 beat per minutes. He is otherwise well.

The <u>long-term</u> management → <u>Statins + Clopidogrel</u>

- ◆ Statins → for all patients with Hx of stroke or TIAs.
- ♦ Clopidogrel (as there is no AF). If there was AF, we would give warfarin or DOAC.
- ◆ Remember, we give Aspirin 300 mg for 2 weeks followed by Clopidogrel 75 mg.

Example (2),

An elderly female with no significant past medical history presents to the A&E with speech disturbance and asymmetric weakness of face and arm. These symptoms started 3 hours ago. CT scan of the head shows no hemorrhage.

 \lor The best **next** step \rightarrow **Alteplase** (Thrombolytics)

"Acute ischemic stroke presenting within 4.5 hrs of the symptoms"

√ If the question asks about the **long-term** drug:

- If no AF → Clopidogrel (Careful!)
- If with AF → Warfarin or DOAC "eg, apixaban" (Careful!)
- + Statins + control HTN.

In ACUTE Ischemic stroke

- **✓** Maintain O₂ saturation, Temperature, Blood Glucose.
- Do CT scan head to confirm that it is an ischemic not hemorrhagic stroke.

If the patient presents within 4.5 hours of the onset of the symptoms \rightarrow Give Alteplase (preferred over streptokinase).

- Vigive Aspirin 300 mg ASAP either orally or rectally (continued for 2 weeks), followed by Clopidogrel 75 mg (long-term) if no atrial fibrillation. If there is associated AF, give DOAC (long-term) instead of clopidogrel.
- **V** If presents > 4.5 hours of onset or time is not given \rightarrow Aspirin 300 mg ASAP.

Here, the **CT** is already done and confirmed **ischemic** (not hemorrhagic) stroke. The patient presents **3 hours** after the onset of the symptoms.

In acute ischemic stroke, what if the time of the onset of symptoms is not given?

→ Start with Aspiring 300 mg (we cannot give Alteplase if > 4.5 hours as it would be useless).

Example (3),

An elderly female with no significant past medical history presents to the A&E with speech disturbance and asymmetric weakness of face and arm. These

symptoms **resolved** in 3 hours ago. CT scan of the head shows no hemorrhage.

 \lor The best **next** step \rightarrow **Aspirin**.

Since the question mentions that the symptoms resolved in <24 hours

- → This is a Transient ischemic attack -TIA- (not a stroke).
- → Give Aspirin (for 2 weeks, then change it to clopidogrel)

(In TIA, we do NOT give alteplase -no thrombolysis in TIA- Be careful)!

→ TIA requires Carotid doppler scanning. (CT angiography of carotid vessels NOT of brain)!

Very Important:

- **If** the question **did not mention that the symptoms have resolved**, and the patient presents within the first 4.5 hours of the onset of the symptoms, and the CT head shows NO hemorrhagic \rightarrow ischemic stroke \rightarrow the answer would be thrombolysis (alteplase).
- The long-term management of ischemic stroke would be:

Statins -atorvastatin- (for all patients, regardless of their lipid profile).

- + Aspirin for 2 weeks followed by clopidogrel for life (if **no** atrial fibrillation).
- (If <u>atrial fibrillation</u> is present, give warfarin or DOAC -eg, <u>apixaban</u>- instead of clopidogrel).
- + Control blood pressure + Lifestyle modification (eg, \sqrt{salt} , fat, no smoking).

Key 10

Broca's VS Wernicke's Aphasia

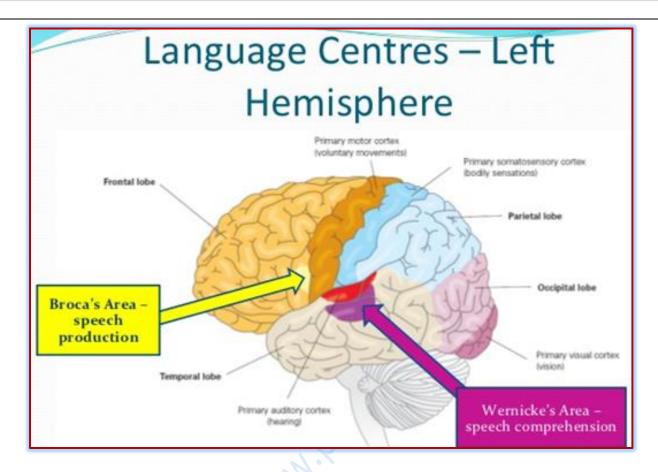
√ Broca's area

→ (in inferior Frontal lobe of the dominant hemisphere- usually the left).

v Wernicke's area

→ (in superior **Temporal** lobe of dominant hemisphere- mostly **left**).

- **▶ Broca's aphasia** → Broken speech. The problem is in <u>speech</u> production, otherwise normal. (non-fluent, slow speech, bad grammar, but they know and understand what they are saying "Good Comprehension").
- ♦ Wernicke's aphasia → What? The problem is with speech comprehension = understanding. (Although fluent speech, they produce sentences that do not make sense, they do not know what they are saying).



Key 11

Rules on Anti-epileptic drugs in Pregnancy

- Before pregnancy → Change Sodium Valproate to another safer antiepileptic drug (such as Lamotrigine [followed by] Carbamazepine)
- [+] Add Folic Acid 5 mg until 12 weeks of pregnancy.
- If she is Already Pregnant but has <u>not been taken</u> any anti-epileptic medications yet (has new onset of epilepsy and Rx is needed)

- → Lamotrigine (first line in pregnancy) OR Carbamazepine (2nd-line).
- If established pregnancy and already taking anti-epileptic medications
- → Do not change the anti-epileptic drug (even if it was Sodium Valproate)!

Sodium Valproate has, comparatively, greater risk of teratogenicity and fetal malformations than Lamotrigine and Carbamazepine.

- If the patient is seizure-free for ≥ 2 years and got pregnant
- → Consider stopping Anti-epileptic drugs.

Remember (imp.):

V Pregnant women on anti-epileptic medications need to take **Folic acid 5 mg** daily (not 400 microgram) until the **12**th week pf pregnancy.

√ If a non-pregnant woman is taking Sodium Valproate to control her seizures, and no alternative is available, advise here to use effective contraception as sodium valproate is very teratogenic.

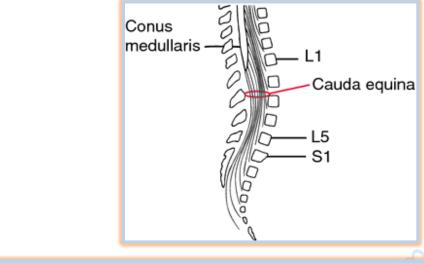
(She needs to avoid pregnancy while on this drug).

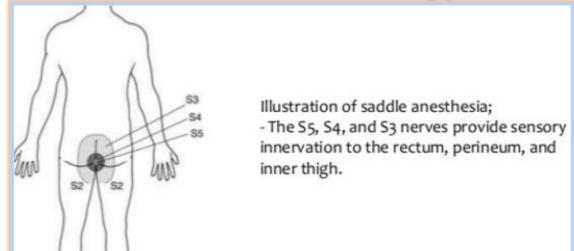
Key Prostate cancer can metastasise to spine causing → Cauda Equina Syndrome
 → Perianal/ groin numbness (Saddle Paraesthesia) Inability to initiate
 voiding "urination" Back pain.

→ Urgent MRI of Spine

Cauda Equina Syndrome

- Cauda equina = bundle of nerves and nerve roots at the lower end of spinal cord.
- It resembles the horse's tail, starts from (T12/L1 to Coccyx).
- Compression of the cauda equina is a surgical emergency!
- Features:
- ◆ Sciatica (pain along the sciatic nerve course: Low back, hips, buttocks, legs).
- ◆ Saddle Paraesthesia (anal/ perianal/ groin numbness).
- ♦ Urinary retention (inability to void).
- ◆ **Fecal incontinence** (inability to control bowel movements, resulting in involuntary soiling).
- The commonest cause \rightarrow *Central Disc Prolapse* that compresses cauda equina.
- It is a surgical emergency.
- Urgent MRI
- Sometimes the answer would be → Urgent referral to orthopaedic surgeon.
- **Urgent Surgical decompression** (to avoid persistent loss of sphincter and motor functions).





In a patient with lower back pain, the presence of (Saddle Paraesthesia) warrants urgent referral to neurosurgical/orthopaedic team for MRI and decompression if needed.

Key 13

Parkinson's Disease:

It is a progressive neurodegenerative condition caused by degeneration of dopaminergic neurons in the **substantia nigra** \rightarrow Low levels of dopamine \rightarrow This results in a classic triad of features: **bradykinesia**,

resting tremors and **rigidity**. The symptoms of Parkinson's disease are characteristically asymmetrical.

■ Features of Parkinson's disease: (BRRP)

- √ Bradykinesia (slow movements).
- **V** Rigidity.
- √ Resting tremors. (present at rest)
- **V Postural instability.**
- **±** Expressionless face.
- Usually, > **65 YO**.
- ◆ The main drugs for Parkinson's Disease -in general-
- → Levodopa, Co-careldopa, Co-beneldopa
- and Cabergoline (Dopamine "D2" Agonists).
- ◆ The main drugs for controlling TREMORS and Dystonia in Parkinson's disease (Parkinson's diseases induced tremors, especially if < 55 YO)</p>
- → Anti-cholinergics (Benzhexol, Orphenadrine).
- ♦ for nocturnal and early morning hypokinesia and rigidity
- → Controlled release preparations.

Important notes on Parkinson's Disease

"Previously asked":

- ✓ If a Parkinson patient on Co-careldopa (Sinemet ®) or Co-beneldopa develops *hallucination*
- → Reduce the dose of Co-careldopa
- Never use Haloperidol or Metoclopramide in patients with Parkinson's:
- The antiemetic used in Parkinson's patients is:
- → Cyclizine, if refractory vomiting, add → Levomepromazine.
- The rapid tranquilizer (in **delirium**, **psychosis**, **agitated** patients) used in **Parkinson's** patients → **Lorazepam**. "Can be given IV or IM in urgent cases".
- The most likely affected anatomical structure in Parkinson's disease is
- → Substantia nigra

	Delirium	Dementia
Onset	• Acute (hours to days)	Chronic (months to years)
	• Fluctuating symptoms over the day	Progressive
Causes	Infections (eg, UTI), elderly, intoxication, dehydration, Metabolic derangement, Drugs (eg, Benzodiazepines)	Eg, Alzheimer's disease, ag
Reversible?	V	X
Attention, Orientation, Consciousness	Impaired	Normal
Hallucinations and illusions	√	Less common (*Visual Hallucinations with Lebody dementia*)
Mood changes	V	٧

Example (1),

A 63 YO \bigcirc is brought to a hospital by her daughter who says that her mother has a drastic behaviour and mood changes for the last 3 days. The sick lady also claims that there are thieves who enter her flat at night.

The likely Dx is \rightarrow **Delirium**

- The key is the abrupt- acute- onset (3 days only).
- + Hallucinations, mood and behaviour changes.
- Dementia is chronic and progressive (over months and years).
- Note, Schizophrenia mainly occurs in early adulthood and it is very rare to be seen in people > 50 YO. Furthermore, auditory hallucinations are much more common in Schizophrenia than visual hallucinations. Also, memory is grossly intact and disorientation is rare.

Example (2),

A 77 YO \bigcirc admitted for UTI for 12 days has developed confusion, fluctuating level of consciousness and disoriented to time and place.

The likely $Dx \rightarrow$ **Delirium**

- (Sudden onset Fluctuating symptoms Infection (UTI) in elderly).
- Remember, UTI in elderly is a common cause of delirium.
- Some features of delirium → Confusion, Mood changes, Disorientation,
 Some memory impairment.

Key 15 A toddler (mainly 6 months – 2 years old) \Rightarrow (\pm Minor injury – pain – fear)

⇒ ± Crying → Loss of consciousness (for less than 1 minute) → Rapid Recovery

V Either → Blue breath holding spells (the young child turns Blue and stops breathing).

V Or → Reflex anoxic seizures (= Reflex asystolic syncope) (= White breath holding attacks) (The young child stops breathing and turns Pale).

- Note, in Reflex anoxic seizures, a toddler is rigid and pale and may have upward eye deviation + Colonic (Jerky) movements. HOWEVER, there is no biting of tongue.
- Reflex anoxic seizures do not cause **Tongue Biting** (an important point to differentiate from it from epilepsy).

- We do not usually need to differentiate blue breath-holding spells from Reflex-anoxic seizures as the management is the same.
- Nonetheless, in the former (blue breath-holding spells), the toddler usually cries vigorously and turns into blue

whereas, in the latter (**Reflex anoxic seizure**), he would attempt to cry followed a **minor injury** (e.g. **falling**) but may not be able to cry, and he turns to **pale**.

■ Management in both cases:

- √ Reassurance.
- $\sqrt{}$ Advice parents to place their child in the recovery position until the episode ends (usually less than 1-2 minutes).
- $\sqrt{\text{Check Ferritin}}$ and treat iron deficiency anemia if present.

Key 16

Some Quick Points on Seizures' types

- A child, ceased breathing → Reflex anoxic seizure/ blue breath holding spells.
- \square Seizure + Fever \rightarrow Febrile seizure.

Seizure can be: Partial (Focal) or Generalized.

 \blacksquare "Partial" (Focal) \rightarrow 1 muscle or 1 group of muscles (one part of the body).

Partial seizure can be:

- V <u>Simple</u> → No LOC "No Loss of Consciousness"
- \lor Complex \rightarrow with LOC.

© Generalized Seizure Important Types:

- **V** <u>Tonic-clonic</u> (Grand Mal Seizure) → Rigidity (Stiffness) followed by jerky movements (Twitches). Tonic means Rigidity (Stiffness). Clonic means Jerky movements (Twitches). There is loss of consciousness during the episode.
- **V** <u>Myoclonic</u> → No rigidity, no LOC, just jerky movements (muscle twitches) mainly for < 1 second up to a few seconds.
- **V** <u>Absence</u> (Petit Mal Seizure) → No aura, <u>Staring</u>. "Sudden loss of awareness for a few seconds. Mainly in children. The child may be staring, lip smacking, eye blinking".

Example (1),

While dancing on a stage, a known alcoholic young man suddenly collapsed and his body went stiff then there was twitching of his legs followed by involuntary voiding. After gaining consciousness, he was drowsy and confused for several minutes before full recovery.

The likely $Dx \rightarrow \overline{\text{Generalised Tonic Clonic Seizure}}$.

- Collapsed = (Loss of consciousness) + his (Body) involved → Generalised
- Went stiff → **Tonic**.
- Twitching → Clonic.
- → Generalised Tonic Clonic.

Example (2),

An 8 YO boy developed a seizure firstly affecting his arm that lasted for several minutes. During the whole episode, he was unconscious and unable to remember the events that led to his seizure. CT scan of head shows a lesion on the left cerebral hemisphere.

The type of seizure \rightarrow Partial Complex Seizure.

The likely cause of seizure \rightarrow **Space-occupying lesion** (seen on the CT).

 \lor Affecting his arm (a part of the body) \rightarrow **Focal** (**Partial**).

 \lor Loss of Consciousness \rightarrow Complex.

V Partial complex means there is something blocking a part of one cerebral hemisphere which is compatible with the CT finding \rightarrow A space occupying lesion.

∨ **Space-occupying lesions** (eg, Abscess, Tumour, Hematoma) can cause headaches, blurry vision, diplopia (CN palsy), papilledema, and also seizures.

V In Huntington's disease → jerky, random uncontrolled movements (Chorea) that start to appear between the age of 35-45 YO.

 \forall In **Epilepsy** \rightarrow There has to be **2 unprovoked seizures with > 24 hours apart**.

Example (3),

A 44-year-old man presents to the ER with a history of involuntary jerking of his right hand that involved his right arm for 4 minutes. He remained conscious and oriented throughout the episode. There is no postictal confusion. He did not have similar episodes before and does not have any history of seizure or any other neurological disorders. What is the most likely diagnosis?

- → Simple partial seizure.
- \checkmark Affecting one part of the body \rightarrow partial (focal) seizure.
- √ No loss of consciousness → Simple.
- → Simple partial seizure.

Key 17

Obstructive Sleep Apnea Syndrome

Features:

- Middle-aged to an elderly male.
- **†** Daytime sleepiness (somnolence) and fatigue.
- Morning headaches.
- Snoring loudly at night (± episodes of apneas).
- Night awakenings with difficulties to get back to sleep.
- Associated with HTN, DM, Obesity, Alcohol consumption.

Diagnosis:

- Initial → Pulse oximetry, Overnight study of breathing patterns.
- Most appropriate (Gold standard) → Polysomnography.

Polysomnography is also called a **sleep study**, is a comprehensive test used to diagnose sleep disorders.

Polysomnography records brain waves, oxygen level in blood, heart rate and breathing, as well as eye and leg movements during the study.

☐ Treatment:

- Conservative → weight loss, stop alcohol.
- CPAP "Continuous Positive Airway Pressure".
- Rarely, surgical alleviation of an obstruction (eg, Adenoidectomy, Tonsillectomy) is needed.

Key 18	Important CNS DDx	Guillain-Barre Syndrome (GBS)	Myasthenia Gravis	Motor Neuron Disease (eg, MS)
	Weakness	٧	٧	٧
	Fatigue	X	VVV "Hallmark"	X
	Reflexes	Absent or ↓	Present	May be 🕇
	Pain	Often	X	X
	Fasciculations	X	X	V
	Others	 ♦ Young age. ♦ Ascending polyneuropathy. ♦ After a trigger (eg, infection) 	 ◆ Tires easily. ◆ D features (Drooping eyelids "ptosis", Diplopia, Dysphonia, Dysphagia, Dysarthria) 	

Important Note:

- The mechanism of **Guillain barre syndrome**
- → Autoimmune degeneration of myelin sheets of the <u>peripheral</u> neurons.

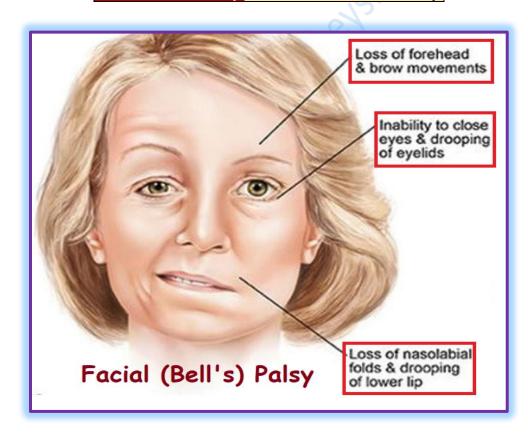
Another valid answer \rightarrow **Demyelination of peripheral nerves**.

✓ Ascending weakness (usually begins in LL) + Loss/ reduced tendon reflex ±
Hx of GIT or Respiratory infection

- → Guillain Barre Syndrome.
- For $Dx \rightarrow Nerve$ conduction study.
- ▼ → To confirm Dx of Myasthenia Gravis
- → Serum <u>skeletal</u> muscle <u>nicotine</u> acetylcholine receptor antibody.

Key 19

Facial Palsy = 7th CN Palsy



In this picture, the affected side is the left side (left facial nerve). She is trying to smile but only her right facial muscles are working. Also, there is loss of

forehead creases on the left side. Note, in addition, her left eye is opened (cannot close it). This is a case of <u>left side</u> facial palsy or Bell's palsy.

- Bell's palsy is not exactly the same as facial nerve paralysis. However, the same nerve is affected in both with similar features. You do not need to know differences.
- Facial palsy can occur due to a viral infection (eg, following a flu).

■ Features of Bell's Palsy (same as CN 7 or Facial nerve palsy):

- **V** Weak facial muscles (eg, weak movement of eye and lips). ■
- V Ptosis and dry eye (on the affected side).
- V Inability to close eye (on the affected side).
- √ Drooping of lower lip (on the affected side).
- V Loss of nasolabial folds (on the affected side).
- V On smiling, the face is drawn across to the opposite side, whereas <u>the</u> affected side will remain frozen.
- V Sometimes, there will be **pain around the same-side ear** but <u>without rash</u> (DDx from Ramsay Hunt Syndrome "Herpes Zoster Oticus" that has painful rash).
- \forall Important \rightarrow in severe cases \rightarrow Dysarthria and difficulty with eating.

■ Treatment: (Important) → Prednisolone (Corticosteroids).

Within 72 days of onset, give high doses of <u>prednisolone</u> for 10 days.

Dysarthria = difficult or unclear articulation of speech that is otherwise linguistically normal.

Note:

- ◆ Facial nerve (7th) → Innervates the voluntary muscles of face (muscles of expression).
- ◆ Trigeminal nerve (5th) → Innervates the involuntary muscles of the face, muscles of mastication and the cornea.

Horner's syndrome → Unilateral Ptosis, Miosis, Anhidrosis.

→ Compression of the Ipsilateral Sympathetic Chain

Trigeminal Neuralgia 5th CN.

Unilateral Electric shock-like pain/ sharp, shooting stabbing pain in one side of the face (unilateral) which is sudden, episodic and lasts for a few seconds to

minutes with recurrence. It might start in the jaw angle and radiate to the temporal region or forehead.

The pain is worsened on chewing, movement or touch.

Rx → Medication is tried first, then surgery

√ Medications examples → Carbamazepine (First line) (Important √)

Others → lamotrigine, phenytoin, gabapentin.

Sometimes, the answer will be (*Anticonvulsants*). Carbamazepine is an anticonvulsant.

V Surgery → microvascular decompression.

Note, in trigeminal neuralgia, corneal reflexes are usually intact.

Key 20 **Status epilepticus** (SE) is a single **epileptic** seizure **lasting > 5 minutes** or two or more seizures within a five-minute period without the person returning to normal between them.

Steps of management of status epilepticus:

■ First Step → 2 separate doses (10-20 minutes in between) of either:

V IV Lorazepam "1st line in-hospital and if there is established IV access". [or]

V Buccal Midazolam [or] (Rectal Diazepam) "If no IV access or in-community"

If 2 separate doses of either (IV lorazepam) or (Rectal Diazepam) or (Buccal Midazolam) have been given but the seizure is still ongoing, move to step 2

Second Step → IV Phenytoin

Important: Phenytoin is preferred over phenobarbital.

Third Step \rightarrow Refer to ICU \rightarrow Intubation, IV Phenobarbital.

Key 21

■ In a suspected stroke:

- ◆ The "initial" investigation → CT scan Brain "To exclude hemorrhage"
- ◆ The "most appropriate" investigation → MRI Brain.

This specifically applies for lesions of the **posterior fossa** (eg, **Cerebellum**) where a patient would present with **Ataxia** (unsteadiness, difficulty walking) and **slurred speech** \rightarrow **MRI** is much better than CT.

Pay attention to the question's words!

MRI = Magnetic Resonance Imaging.

Key 22

Normal Pressure Hydrocephalus (NPH)

Hakim's triad (GDU)

V Gait abnormality (Ataxia, Waddling gait, imbalance walking)

V Dementia (Behavioural, Cognitive changes, Forgetful) "Reversible type of dementia".

V Urinary Urgency ± incontinence.

+ **ELDERLY**

Wet, Wobbly and Wacky Grandpa

Wet → Urinary incontinence.

Wobbly → Gait abnormality.

Wacky → Dementia (behavioural changes)

Grandpa → Old man.

 $Dx \rightarrow$

∨ Clinical triad + CT/ MRI brain → Enlarged lateral and third ventricles.

√ Lumber infusion test (intrathecal infusion test): A New NICE option.

Next step → Perform lumbar puncture and CSF pressure monitoring.

$Rx \rightarrow$

CSF Shunt (e.g. Ventriculoperitoneal, Ventriculopleural or Ventriculoatrial shunt).

Key 23

	Epilepsy	Non-Epileptic Attack Disorder (NEAD)			
	Genetic Factors	Hx of child abuse (either sexual or physical)			
Duration of the episode	Less than 2 minutes	More than 2 minutes			
Pelvic Movement	X	V			
Asynchronous movements	X	V			
Eye	Open	Closed			
If eye is closed	Easy to open manually	Difficult to open			
		manually			
Drooling of saliva	٧	X			
Tongue biting	٧	X			
Self-injury during attack	٧	X			

Urine incontinence	٧	X
Post-ictal confusion	٧	X
Post-ictal EEG	Slow	Normal

IMPORTANT: To diagnose **NEAD** or if **in doubt** of the Diagnosis

→ Video EEG (Video Electroencephalogram)

Note, Video EEG can also be beneficial to rule out epilepsy (e.g., in a patient with conversion disorder who has attacks of inability to talk but they do not lose consciousness).

Important, if "**refer to first-fit clinic**" was among the options and the features are **suggesting epilepsy** (e.g., tongue biting, 2 minutes, post-ictal confusion) pick "refer to first-fit clinic". There would be neurologists who will take detailed history and give a management plan.

Key Take A Rest 😝

Key Important Tremor Types 25

• Essential tremors → symmetrical tremors, initially intermittent but over time they become persistent, absent at rest and during sleep, do not resolve with distraction. Essential tremors improve with alcohol consumption. Voluntary movements worsen tremors (This presentation is called kinetic tremors), and outstretching hands (when hand is held against gravity) worsens tremors also (this is called postural tremor).

- Parkinson's tremors → present at rest. (+ others, eg, bradykinesia, rigidity). Starts on one side. (Parkinson's tremors are more obvious at rest whereas essential tremors are more obvious on movements).
- **Psychogenic tremors** → absent at rest, **resolves** with distraction.
- Cerebellar tremors → intentional tremors (eg, when trying to voluntarily catch something or touch his nose) + other cerebellar features (eg, nystagmus, ataxia, dysarthria).
- Alcohol-dependence (withdrawal) tremors → seen in chronic alcoholics who suddenly stop drinking alcohol. They are usually postural tremors also. Usually associated with anxiety, sweating, hyperactivity.

Key A patient with liver carcinoma (Or Breast Cancer) that is metastasised to Brain presents with generalised muscle weakness, Nausea, Vomiting, **Polyuria** and **Polydipsia**.

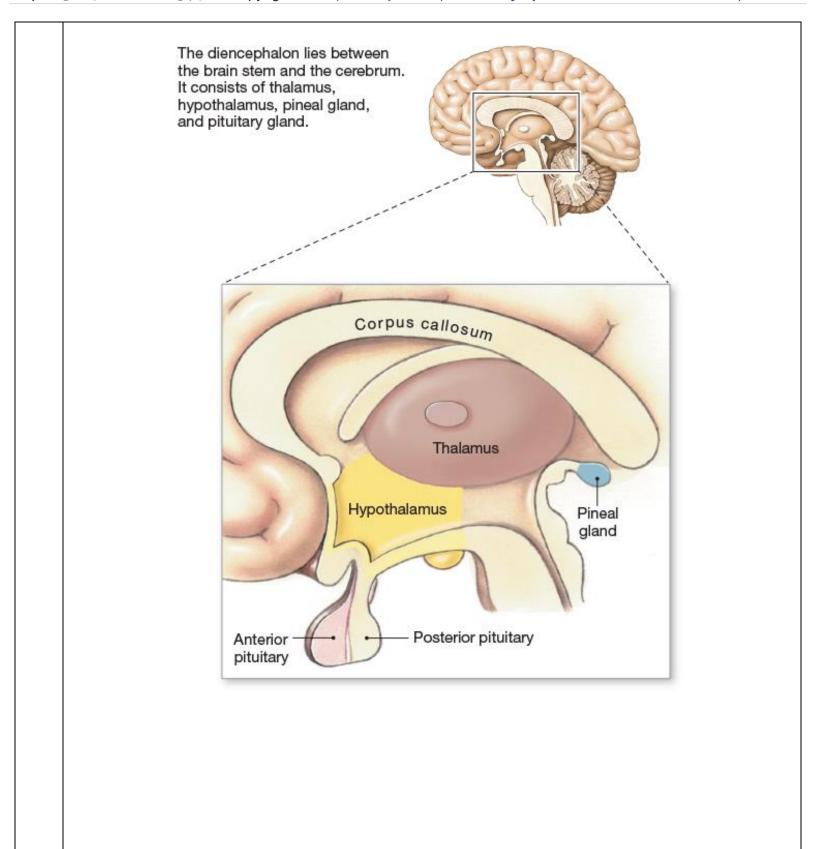
The likely site of the brain lesion "Metastasis" → Diencephalon

As there is **polyuria** + **Polydipsia** with Hx of **brain lesion** → think of Cranial (central) Diabetes Insipidus (DI).

Remember, in Diabetes insipidus $\rightarrow \uparrow$ Na+ (hypernatremia), whereas in SIADH \rightarrow dilutional hyponatremia.

- ♦ DI → DIencephalon.
- ♦ SIADH → Cerebrum/ Cerebellum.

- Diencephalon → (Thalamus, **hypothalamus**, epithalamus, subthalamus and Posterior pituitary).
- **Cranial diabetes insipidus** is a condition in which the hypothalamus does not produce enough anti-diuretic hormone (ADH).
- **Nephrogenic diabetes insipidus** is a condition in which the kidneys fail to respond to anti-diuretic hormone.



Key 27

Comparison of Bulbar and Pseudobulbar palsy

Bulbar Palsy	Pseudobulbar Palsy
Disturbance to X, XI, XII, sometimes VII, rather than the corticobulbar tracts (LMN)	Degeneration of corticobulbar pathways to V, VII, X, XI, XII (UMN)
lower motor neurone signs present	lower motor neurone signs absent
gag reflex (-) bovine cough	gag reflex (+/n)
wasted tongue, fasciculations	spastic tongue
jaw jerk (n)	jaw jerk (+)
nasal speech	spastic dysarthria
normal emotions	labile emotions
signs in limbs	bilateral UMN

+ = increased; - = reduced; n = normal

Bulbar area → An area of the brain composed of the **cerebellum**, **medulla** and **pons**. (Basically, the bulbar region is made up of the brainstem minus the midbrain and plus the cerebellum). The bulbar region is responsible for many involuntary functions that keep us alive.

Example,

A 66 YO \bigcirc presents with difficulty in swallowing, aspiration pneumonia, **bovine cough**, **wasted tongue** that sits in the mouth and with **fasciculations**, difficult to articulate certain words, nasal regurgitation of food.

The likely cause of her dysphagia \rightarrow **Bulbar Palsy**.

- ♦ Note, the phrase "bovine cough" is used to describe the non-explosive cough of someone unable to close their glottis. It is seen in vagus nerve lesions, and may be associated with dysphonia.
- Wasted tongue with fasciculations
- Bovine cough (in ability to close epiglottis)

These are seen in **Bulbar palsy** (not pseudobulbar palsy).

Key 28

- Multiple Cranial nerve affection (e.g. Facial numbness "Trigeminal nerve" + Diplopia and Ptosis "Oculomotor nerve")
- → Brainstem lesion

Oculomotor (3rd CN) → Midbrain

Trigeminal (5th CN) \rightarrow Pons

Key 29 **□ Ipsilateral oculomotor nerve palsy (e.g.** left eye ptosis, left mydriasis -dilated pupil-, left eye is deviated outwards and downwards)

[+] Contralateral hemiparesis (e.g. right arm + right Leg)

- → Weber's Syndrome (MIDBRAIN infarct) (PCA block: Posterior Cerebral Artery)
- Ipsilateral Horner's syndrome + Loss of Pain and Temperature sensation in Face
- [+] Contralateral loss of Pain and Temperature sensation in Limbs.
- → Wallenberg's Syndrome = Lateral MEDULLARY Syndrome (PICA Block: Posterior Inferior Cerebellar Artery).

Example 1,

A 63 YO \bigcirc with uncontrolled HTN presents with right hemiparesis. Her left pupil is dilated. Her left eye shows ptosis and is deviated outwards and downwards.

The likely affected area → Midbrain

- Ipsilateral oculomotor nerve palsy.
- [+] Contralateral hemiparesis.
- → Weber's syndrome "Midbrain infarct".

Example 2,

A 66 YO \bigcirc with uncontrolled HTN presents with dysphagia, ataxia. She also reports a loss of sensation on the right side of her face and a loss of sensation on her left leg.

The likely affected area \rightarrow Lateral Medulla.

- Loss of sensation on right side of face. (**Ipsilateral** Loss of sensation in the **face**).
- Loss of sensation on Left leg. (Contralateral Loss of sensation in trunks and limbs).
- → Lateral Medullary Syndrome = Wallenberg's syndrome.

Key 30

Cranial Nerves Nucleus:

 \vee 1, 2 \rightarrow Cerebral Cortex

 \vee 3, 4 \rightarrow Midbrain

 $\sqrt{5}$, 6, 7, 8 \rightarrow Pons

 \vee 9, 10, 11, 12 \rightarrow Medulla

2-2-4-4

Key 31

Horner's syndrome → Ptosis, Miosis, Anhidrosis.

• Horner's syndrome is a rare condition characterized by <u>miosis</u> (constriction of the pupil), <u>ptosis</u> (drooping of the upper eyelid), and <u>anhidrosis</u> (absence of sweating of the face).

• It is caused by damage to the <u>sympathetic nerves</u> of the face. The underlying causes of Horner's syndrome vary greatly and may include a tumor, stroke, injury, or underlying disease affecting the areas surrounding the sympathetic nerves.

Key 32

- Symptoms of (Cerebellar Lesion) e.g. ataxia -unsteadiness-, nystagmus, slurred speech
- [+] Cranial Nerves Symptoms (e.g. Vertigo: 8th CN Vestibulocochlear nerve Diplopia: 3rd CN Oculomotor nerve)
- → Brain Stem lesion
- ♦ Logically, it cannot be a lesion of cerebellum only.
- ♦ Cerebellar lesion does not cause cranial nerve symptoms. However, Cranial nerves problem can cause similar features to cerebellar lesion's features + Other CN features.
- ◆ Cerebellar lesion → Ataxia Intentional Tremors Dysarthria Nystagmus No vertigo, ptosis, diplopia, facial numbness (these are Cranial Nerve symptoms)
- ♦ Important, some may pick cerebellopontine angle tumor thinking it is Schwannoma (Acoustic Neuroma). Remember:

DVT (Deafness, Vertigo, Tinnitus) + CN Palsy → Acoustic Neuroma. (There should be hearing loss, tinnitus)

Key Alcohol:

UK guidelines recommend that a person should drink

- No more than 14 units a week,
- No more than 3 units a day,
- with at least 2 alcohol-free days a week.
- A patient is admitted for hemoptysis. After 24 hours while in hospital, he develops sweating, tremors, alternating levels of consciousness, temp of 37.3. He has been drinking alcohol every day for the last year.
- The likely Dx → Acute Alcohol Withdrawal
- The most appropriate $Rx \rightarrow \frac{\text{Chlordiazepoxide}}{\text{Chlordiazepoxide}}$. i.e. Benzodiazepines.
- What is next to be added to chlordiazepoxide? → Thiamine -Vit. B1-
- "To prevent Wernicke's encephalopathy".
- Sometimes the answer would be the drug family → Benzodiazepines.

- Sometimes the answer, if there are withdrawal **seizures**, is \rightarrow **IV Lorazepam**
- Or **Diazepam** "If IV Lorazepam is not in the options)
- ♠ Remember, if an alcoholic patient presents with CAS (Confusion, Ataxia, Squint: Ophlamoplegia, Nystagmus) → Give Vitamin B1 (IV Thiamine) as this is likely Wernicke's encephalopathy.

Management of Acute Alcohol Withdrawal: (Important $\sqrt{\ }$)

- ♦ Benzodiazepines √
- \forall First line \rightarrow Chlordiazepoxide (benzodiazepines).
- √ First line if there is withdrawal **Seizure/ hallucination (delirium tremens)**
- → Lorazepam (Or Diazepam "If IV Lorazepam is not in the options).
- ◆ Vitamin B1 (Thiamine) = (IV Pabrinex): To prevent Wernicke's encephalopathy.

Other drugs related to Alcohol Intake:

- ◆ **Disulfiram**: Promotes Abstinence. (Serves as a **deterrent** when he takes alcohol).
- ◆ Acamprosate: Reduces Craving.

Note that if withdrawal symptoms continue, it can advance to **Delirium Tremens** (Hallucinations that are indistinguishable from reality and associated with severe confusion, tremors and disorientation)

In summary: (Commonly asked)

- ✓ Acute alcohol withdrawal (12-24 hours after stopping alcohol: sweating, tremors, altered mentation, ± Hallucination
- → Chlordiazepoxide "First" + Thiamine (Vit. B1)
- v If with "seizure/ hallucinations" → IV Lorazepam. Or Diazepam "If IV Lorazepam is not in the options)
- Wernicke's encephalopathy (CAS: Confusion, Ataxia, Squint: ophthalmoplegia, Nystagmus, diplopia), may present 12-24 hours after stopping alcohol as well.
- → IV Vitamin B1 (Thiamine) (IV Pabrinex)
- or (High potency Vitamin B Complex).

- ✓ An alcoholic wants a medication to serve as a <u>Deterrent</u> when he takes alcohol "Abstinence" \rightarrow <u>Disulfiram</u>.
- ✓ An alcoholic wants a medication to reduce his **Craving** for alcohol
- \rightarrow A<u>c</u>amprosate.
- ✓ An alcoholic wants a medication to help <u>reduce</u> withdrawal symptoms
- → Chlordiazepoxide.

Key Again, to simplify:

34

■ Muscle weakness [+] Loss/ Reduced reflexes [±] Hx of GIT or Respiratory infection → Guillain Barre Syndrome.

Do nerve conduction study.

- Fatigue, Fatigggue, Fatiggggue + Muscle weakness + Normal Reflexes + "D features: Diplopia, Drooping eyelid, Dysphonia, Dysphagia)
- → Myasthenia Gravis
- Weakness + (INCREASED) reflexes + Fasciculations
- → Motor Neuron Disease (MND).

Key 35 ■ Fatigue, Fatigue + Muscle Weakness + Normal reflexes ± **D** features (Drooping eyelids "ptosis", Diplopia, Dysphonia, Dysphagia, Dysarthria)

→ Myasthenia Gravis

- Painless muscle weakness that worsens with exercise, Tires easily Speech fades, Difficulty climbing stairs and reaching for items on shelves, Difficulty chewing and swallowing, Diplopia, Normal reflexes ± FHx of an autoimmune disease (e.g. hypothyroidism)
- → Myasthenia Gravis
- → To confirm Dx
- → Serum skeletal muscle nicotine acetylcholine receptor antibody
- ◆ Emphasis of Fatiguability (Tires easily)
- ♦ All muscles are weak leading to *diplopia*, *dysphagia*, *dysarthria*, *dysphonia*, *drooping eyelid*
- ◆ Normal finding on examination (normal intact reflexes).
- ◆ Association with another autoimmune disease (e.g. thyroid disease).
- Key A vasovagal syncope → sudden hypotension, usually due to overwarm
 environment, Excitement, Pain, Fear, Prolonged standing period, or after visual stimuli e.g. seeing blood. The patients usually feel dizzy and "tunnel vision" before the attack but not always.
 - ♦ Brief LOC "Loss of conscious" with rapid recovery.
 - ♦ Hint → YOUNG FEMALE, No chest pain, No palpitation, Normal ECG

Key 37	Polycystic Kidney Disease → Berry Aneurysm → Subarachnoid Hemorrhage	
Key 38	NAD → Nystagmus, Ataxia, Dysarthria → Cerebellar lesion	
Key 39		
	This present as peripheral paraesthesia (Tingling/ Numbness/ Burning	
	sensation of fingertips and toes, hands and feet) in a glove-and-stocking distribution.	
	Sometimes: Sharp, Shooting, Electric-shock-like pain. [a] A walk to Remember:	
	After chemotherapy, any of the following may occur:	
	 Tumour Lysis Syndrome → [UK Pc: ↑ Urate (Uric Acid), ↑ K+, ↑ Phosphate, ↓ Ca++] 	
	Febrile Neutropenia (Fever, very low Neutrophils) → immediate IV Abx.	
	Chemotherapy-induced peripheral neuropathy	

 \rightarrow peripheral paraesthesia – glove-and-stocking distribution-, or

sharp shooting peripheral pain.

Important S/E of Vincristine (Chemotherapy):

- Chemotherapy-induced peripheral neuropathy (due to neurotoxicity).
- SIADH → dilutional Hyponatremia.

Key 40 If a patient presents with features of stroke of cerebellum and/or brainstem (e.g., *nystagmus*, *ataxia*, *vertigo* -*Dizziness*-, *dysdiadochokinesia*), the site of the stroke is in

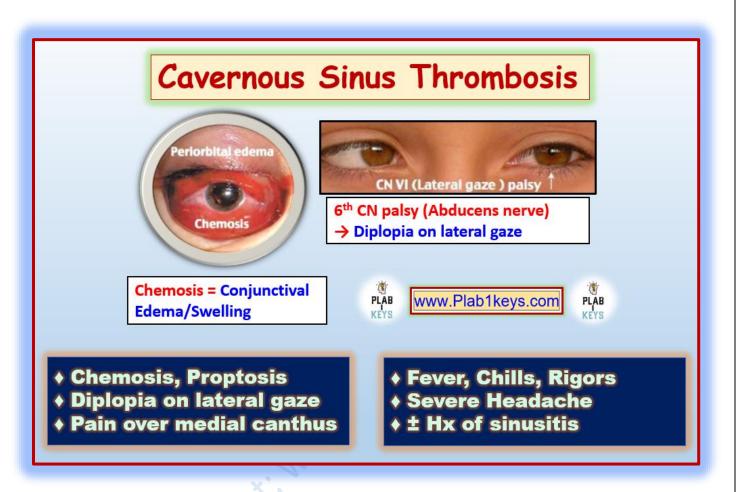
→ the **Posterior Circulation**

Request → MRI of the head "not CT"

- <u>Dysdiadochokinesia</u> (DDK) → impaired ability to perform rapid, alternating movements. Complete inability is called <u>Adiadochokinesia</u>.
- Dysdiadochokinesia is a feature of cerebellar ataxia

Key 41

Cavernous Sinus Thrombosis



Key A 40 YO chronic alcoholic with established liver disease is brought to hospital
 after a heavy drinking episode. He is unable to walk straight, complaining of double vision, and is shouting obscenities and expletives (saying bad words).

The likely $Dx \rightarrow Wernicke's encephalopathy$.

Remember, **CAS** (in *chronic alcoholic* or *persistent vomiting* e.g. in pregnancy)

- Confusion (saying bad words = not fully conscious)
- Ataxia (unable to walk straight)
- Squint (Diplopia, Ophthalmoplegia)

The $Rx \rightarrow IV$ thiamine (Vitamin B1) = IV Pabrinex

Key 43

Frontotemporal Dementia (Pick' Disease)

■ If you see some of the following features, think of Pick's disease (FTD):

- ♦ Making <u>sexual</u> or <u>inappropriate</u> comments (*Disinhibition*). (√)
- → Orbito-frontal lobe problem. (or frontal, or fronto-temporal).
- ♦ Struggling with word choices. (\checkmark) → *Temporal lobe*.
- ♦ Loss of social interest (disengagement). (√)
- ♦ Acting inappropriately or impulsively. (♥)
- ♦ Personality and behaviour changes. (√)
- ♦ Neglecting personal hygiene. "Not grooming themselves" (V)
- ♦ Over-eating.
- ♦ Insisting on ingesting sweet things.
- ♦ Appearing selfish or unsympathetic.
- ♦ Loss of motivation.

- Loss of inhibition (Disinhibition) is characteristic for Frontotemporal dementia.
- Disinhibition → Socially or contextually inappropriate nonaggressive verbal, physical, and sexual acts that reflect a lessening or loss of inhibitions and/or inability to appreciate social or cultural behavioural norms.

e.g., inappropriate/ sexual comments, Urinating on the sofa

- ♦ Focal gyral atrophy with a knife-blade appearance is characteristic of Pick's disease.
- ♦ Macroscopic changes → Atrophy of the frontal and temporal lobes.

Example,

A 77 YO or was brought to a memory clinic. He was noted to be disengaged, expressing boredom and making inappropriate comments to the doctor of a sexual nature.

→ Frontotemporal Dementia (Pick's disease)

The affected anatomical structure \rightarrow Orbito-frontal lobe.

Remember

■ OLD man + GDU (Gait abnormality/ Dementia -behaviour changes-/ Urine incontinence) = Wet, Wobbly, Wacky Grandpa

→ NPH (Normal Pressure Hydrocephalus).

Next step → Perform lumbar puncture and CSF pressure monitoring.

Then \rightarrow CSF Shunt.

Key 44

Contralateral Paralysis and Sensory Loss (Face and Arm)

- + Ipsilateral Gaze Preference
- → Middle Cerebral Artery (MCA) → supplies the lateral part of the hemisphere.

Other features → Aphasia, Homonymous hemianopsia.

Example (1),

An elderly \circlearrowleft with Hx of MI presents with left arm paralysis, sensory loss on the left side of the face, right-sided gaze preference and homonymous hemianopsia.

The likely affected artery → Right Middle Cerebral Artery.

- ♦ Anterior cerebral a. → Frontal and medial cerebrum.
- ♦ Middle cerebral a. → Lateral part of the hemisphere.
- ◆ Posterior cerebral a. → Occipital lobe.

♦ Basilar a. → Cerebellum, Brainstem, Occipital lobe.

Example (2),

A 73 YO \bigcirc is brought from the nursing home as she complains of sudden onset of **right** hemiplegia, homonymous hemianopia and dysphasia. O/E, brisk reflexes and clonus are noted.

The likely affected vessel \rightarrow <u>Left Middle Cerebral artery.</u>

Key 45

Alzheimer's disease

Alzheimer's disease (AD) is a progressive neurodegenerative disease of the brain accounting for the majority of dementia seen in the UK and in the world.

- The strongest genetic risk factor for Alzheimer's disease (AD)
- → APOE ε4 gene

Clinical Features of Alzheimer's Disease:



Loss of memory



Language problems



Difficulty in doing simple tasks



Disorientation in time and space



Loss of reasoning capacity



Difficulty in having elaborate thoughts



Loss of objects



Mood changes



Behavioural changes



Loss of initiative

Source: fpmaragall.org

- ♦ Elderly (> 65 YO).
- ◆ **Early** → Recent (Short) memory loss, then distant but immediate recall is usually intact Difficulty finding words.
- ◆ After some time → Confusion Inability to do simple or usual tasks (eg, cooking) Inability to make decisions Progressive language deficits.
- ◆ Late → Disorientation (eg, getting lost easy) Behavioural changes Death

Diagnosis (not very important)

- MRI Brain
- HMPAO, SPECT "Single photon emission CT"
- → (to differentiate Alzheimer's from Pick's disease -Frontotemporal dementia-)
- MMSE "Mini Mental State Exam" → To assess the severity of cognitive impairment.

MMSE full score is 30:

25-30 → Normal \blacksquare 21-24 → Mild \blacksquare 10-20 → moderate \blacksquare < 10 → Severe

Note, normal MMSE does not exclude the Dx of Alzheimer's.

Management of Alzheimer's Disease

(Important to memorise all these drugs)

♣ Acetylcholinesterase Inhibitors:

√ Rivastigmine → First line (mild to moderate cases).

√ Others: **Donepezil**, **Galantamine**.

♣ Memantine (NMDA-receptor antagonist) → Second line (or in severe cases, suitable for patients with LBBB, RBBB, bradycardia, heart block).

Memantine is also safe in Alzheimer's patients with LBBB (M shaped or notched R wave in V6). Important $\sqrt{}$.

Example (1),

A 65 YO \bigcirc has become increasingly distressed and cries all the time. She has been forgetting to lock her doors and to turn off the stove. She, for the first time, has forgotten her son's birthday this year. She easily gets lost when she goes out. Sometimes, she puts her keys in the washing machine. She has become unable to use her microwave to cook food anymore. Her recent memory is impaired; however, her immediate recall is intact. She scored 26/30 on the MMSE.

The likely $Dx \rightarrow Alzheimer's disease$.

■ Forgetful elderly (forget to look doors, forget birthdays, forget names of people and places), easily getting lost (Disorientation), unable to do simple tasks (eg, cooking)

→ Alzheimer's disease.

Example 2,

An 80 YO man is brought by his daughter to a clinic. He is having difficulties in remembering what his daughter has told him earlier in the day. He is being more forgetful. He repeats himself in a conversation and finds it difficult to express himself. His MRI scan shows diffuse cortical atrophy. His ECG shows prolonged PR interval. His pulse rate is 50. What is the most appropriate medication to prescribe?

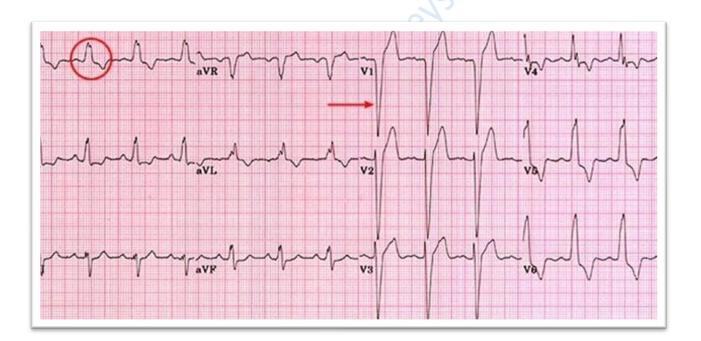
→ Memantine

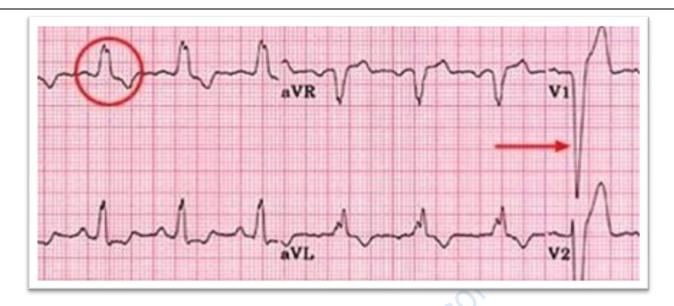
He has **Alzheimer's disease** (symptoms and CT scan suggest Alzheimer's).

Acetylcholinesterase inhibitors (which are first-line in Alzheimer's) such as rivastigmine and donepezil are not suitable in heart conditions as they may lead to pronounced bradycardia and heart block "prolonged PR intervals". In such cases, **memantine** is an alternative.

Example 3,

A 78-year-old man is brought by his daughter to a clinic. His daughter is concerned that her father has been having difficulties in remembering recent events. He has become more confused and disoriented. He is being more forgetful. He repeats himself in a conversation and finds it difficult to express himself. His MRI scan shows diffuse cortical atrophy. His ECG is shown below. Blood pressure is 130/80 mmHg. What is the most appropriate medication to prescribe?





→ Memantine

He has **Alzheimer's disease** (symptoms and MRI scan suggest Alzheimer's). The ECG shows left bundle branch block (LBBB): M notch.

Acetylcholinesterase inhibitors (which are first-line in Alzheimer's) such as rivastigmine and donepezil are not suitable in heart conditions as they may lead to pronounced bradycardia and heart block "prolonged PR intervals".

In heart conditions (eg, LBBB, RBBB, Bradycardia, heart block), **memantine** is an alternative.

Key 46 ■ In Mechanical lower back pain (eg, occurs after lifting heavy objects, intense work, the best advice is

→ Continue "or increase" mobilisation + Take regular analgesia

Bed rest is not advised/ analgesia alone is not sufficient.

- Acute onset back pain elicited after repeated contraction and is **relieved** with movement and changing position
- → Myofascial pain syndrome
- **e.g.** A 55 YO \supseteq present with an acute onset back pain following a long rough car journey. On bending forward, the pain is relieved.
- Key Parkinsonism (Parkinson disease features: Bradykinesia "slow movements" + Resting tremors + Rigidity + Postural instability "Ataxia")
 - → Parkinson's Disease.
 - Parkinsonism [+] Urinary incontinence, Erectile Dysfunction [±] Postural Hypotension "Frequent falls"
 - → Shy-Drager Syndrome.

Mnemonic → "He is *Shy* as he wets his pants, has erectile dysfunction and *Drags* his feet because of his ataxia".

■ Parkinsonism [+] Dementia (Cognitive impairment) [+] Visual Hallucinations ± Delusions

→ Lewy Body Dementia.

Key 48

Lewy body Dementia

- ◆ **Dementia** (the usual presenting feature) with **memory loss** and ↓ **problem solving ability.**
- ♦ Fluctuating levels of awareness and attention.
- ♦ Mild Parkinsonism (e.g. Rigidity, Tremors, Ataxia, Falls)
- ♦ Visual Hallucination (e.g. humans, animals) and illusions. (Important √).
- → Request MRI brain (of choice in Lewy body dementia).

Followed by → **SPECT** (Single-photon emission computes tomography)

That is also known as (DatSCAN) ie, Dopamine transporter uptake imaging.

- ♠ Parkinson's features + Visual hallucinations + Dementia
- → Lewy Body Dementia.
- ♠ Parkinson's features + Urinary incontinence + Postural hypotension
- → Shy-Drager Syndrome.

- ◆ Old man + Gait abnormality + Dementia + Urinary incontinence [GDU]
- → Normal Pressure Hydrocephalus.
- ◆ Old + Making sexual or inappropriate comments (*Disinhibition*) + Loss of social interest (*disengagement*) + Acting inappropriately or impulsively + Personality and behaviour changes ± Over-eating
- → Frontotemporal dementia (Pick's disease).

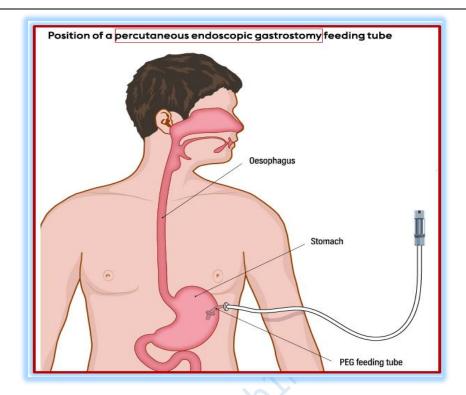
Key A 50 YO ♀ known to have motor neuron disease (MND) with progressive difficulty in swallowing, drooling of saliva, inability to eat properly and sometimes chocks when eating.

The best method for nutrition in this patient

→ Percutaneous endoscopic gastrostomy (PEG).

√ Note that motor neuron disease is a CHRONIC degenerative condition; thus, he requires a LONG-TERM feeding solution.

- **Long-Term feeding** → **PEG** "Percutaneous Endoscopic Gastrostomy feeding tube".
- **Short-Term feeding** → **NGT** "Nasogastric Tube".



Key Do you remember Myasthenia gravis? Fatigue, weakness, NORMAL reflexes, Dfeatures (Diplopia, Dysphagia, Drooling eyelid)?

■ If a presentation similar to Myasthenia Gravis but with (↑) reflexes (+ve for upper neuron signs) + normal autoimmune panel

→ Amyotrophic lateral Sclerosis. (ALS)

The autoimmune panel is (Positive) in Myasthenia Gravis.

The Deep tendon reflexes are Normal in Myasthenia gravis.

Example,

A 56 YO \circlearrowleft presents with a bilateral progressive weakness of his arms (unable to lift heavy objects above his head. SOB while going up the stairs. Hoarseness. Difficulty swallowing liquids. Atrophy and weakness of the muscles of trunk, back, neck and tongue. Deep tendon reflexes are +ve for Upper motor signs. Autoimmune panel is normal.

→ Amyotrophic lateral Sclerosis. (ALS)

Quick Comparison

- Amyotrophic lateral sclerosis (ALS) "a Motor Neuron Disease"
- → Motor weakness (progressive dysphagia and complete paralysis later)
- + **Normal** autoimmune panel + Deep reflexes are (个) **positive** for upper neuron disease.
- Myasthenia Gravis → Similar to (ALS) but with Positive autoimmune panel
- + Normal deep reflexes. (usually eyes are affected; Diplopia, Drooping eyelids)
- Lambert-Eaton Syndrome (Associated with tumours e.g. Lung cancer)

The same presentation as Myasthenia gravis **but** with the following **differences**:

- √ The reflexes are absent and elicited after exercise.
- √ ↑ strength/ power of the weak muscles after repeating the test.

• Multiple Sclerosis → Disseminated in time and place (Recurrent attacks but at different locations) + Associated with OPTIC NEURITIS.

Example: left hemiplegia + Loss of vision and painful right eye with a swollen optic disc "suggesting optic neuritis) + Hx of attacks of hemiparesis/weakness at different limbs.

- Guillain Barre → Hx of GI or Resp. infection + Weakness + ↓ reflexes.
 (Motor "ascending pattern weakness", Sensory and reflexes are all affected)
- Syringomyelia → Motor, sensory, reflexes are all affected but with typical LOSS OF PAIN AND TEMPERATURE (due to affection of Spinothalamic Tract) (e.g. one may burn his fingers without realising as he lacks Pain and Temp sensation).
- Syringobulbia → the same as Syringomyelia but [+] CN involvement (e.g. Facial Palsy]. This is because the Syrinx "the fluid-filled tubular cyst" extends superiorly to involve the brainstem.

Key 51 After a car accident, a young man has become paraplegic. He was **agitated** and would cry every day during the first 2 weeks after the accident while in hospital. He is **unable to remember that accident at all** and refusing to talk about it.

The likely $Dx \rightarrow \overline{\text{Organic brain damage}}$.

√ In Organic Brain Damage → MAC → Memory loss + Agitation + Confusion

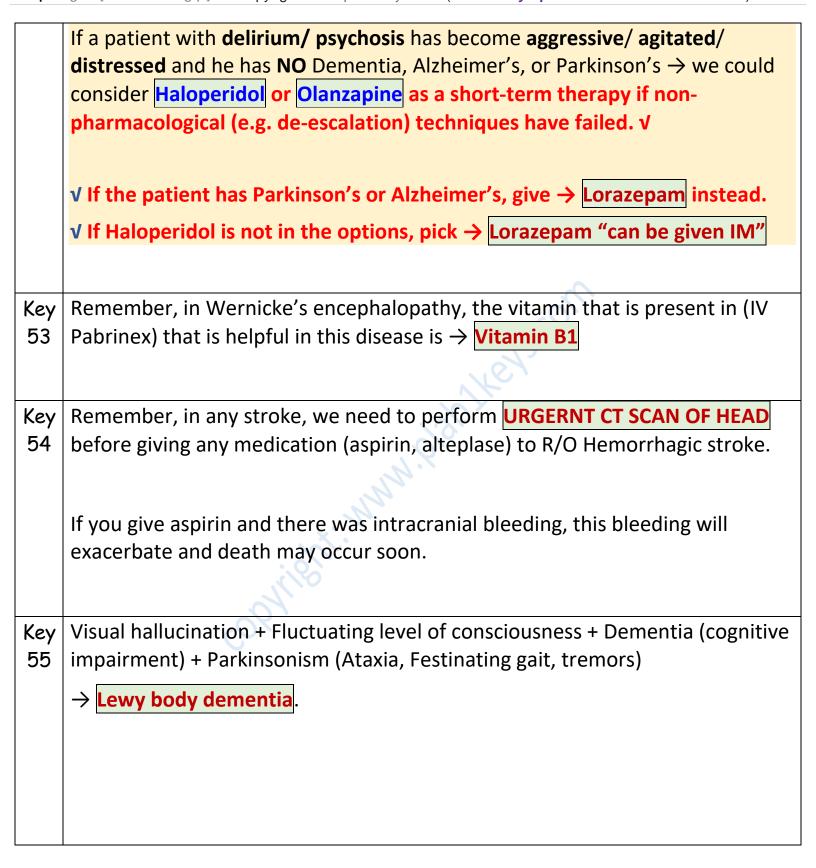
√ This is <u>not</u> a case of <u>Post-traumatic stress disorder</u> (**PTSD**). In (PTSD), it is quite the opposite. The patient <u>would remember</u> the accident with <u>flashbacks</u> and <u>nightmares</u>. Here, he cannot recall the whole accident at all.

V This is <u>not</u> a case of <u>severe depression</u>. It is normal to have sadness after an accident. However, in SEVERE depression, the symptoms would interfere with function. If the severity of sadness persisted + low mood + a degree of functional impairment and disability \rightarrow it would then be Severe depression.

Key An elderly with **Parkinson's** disease has developed an episode of **acute psychosis** and become aggressive. He punches everyone approaching him in the face.

The most appropriate immediate $Rx \rightarrow Lorazepam$ (for rapid tranquilization)

- Lorazepam is a rapid acting benzodiazepine. (Could be given IM here).
- **Haloperidol** (**T**ypical Anti-psychotic) is contraindicated in people with dementia, Alzheimer's, or Parkinson's disease patients.
- Olanzapine and Risperidone (Atypical Antipsychotics) can exacerbate Parkinson's disease.



Key 56

Restless leg syndrome (Willis-Ekbom Syndrome)

- **V** Funny sensation (as insects crawl over legs) aching, tingling, sometimes pain over legs especially at night [+]
- √ Urgency to move the legs (moving legs relieves the symptoms)
- → Restless leg syndrome
- → Check iron (ferritin):
- ♦ If ferritin is low → Give iron supplements (even if Hb is normal, what matters is ferritin).
- **♦ If ferritin is normal** → Give **Dopamine agonist**.
- Note, Restless Leg Syndrome does not usually require treatment. Advice to avoid alcohol, smoking, caffeine, sleep well, exercise is usually enough.
- Dopamine Agonists examples → Ropinirole, Pramipexole, Rotigotine.
- Drugs other than Dopamine agonist that could be used in restless leg syndrome → Gabapentin, Pregabalin (Lyrica®).

Key 57

■ Motor, sensory, reflexes are all affected but with typical LOSS OF PAIN AND TEMPERATURE (due to affection of Spinothalamic Tract) (e.g. one may burn his fingers without realising as he lacks Pain and Temp sensation).

→ Syringomyelia

- **■** the same as Syringomyelia but [+] **CN involvement** (e.g. **Facial Palsy**).
- → Syringobulbia.
- ♦ Other possible features in both cases:

In the upper limbs \rightarrow Absent reflexes. In the lower limbs \rightarrow Hyperreflexia and \uparrow Tone.

In Syringomyelia:

- → A Syrinx (a fluid-filled sac) developed within the spinal cord → Compresses "Spinothalamic tract; thus, leading to loss of temperature and pain sensation
- → The cyst keeps expanding → Compresses other tracts (e.g. Lateral corticospinal tracts, dorsal columns) leading to more features such as muscle wasting, weakness, absent reflexes…etc.
- → When this syrinx extends to the brainstem → features of CN "Cranial Nerves" damage develop such as facial paralysis. The condition is then named "Syringobulbia".

Key 58 **Important**

DEMENTIA	DEPRESSIVE PSEUDODEMENTIA
Progressive onset	Rapid onset
Long term symptomatology	Short term symptomatology
Mood variations	Consistently depressed mood
The patient tries to answer to the questions	Short answers like "I don't know", negativism
Patient is concealing amnesia	Highlighting amnesia
Constant cognitive decline	Fluctuating cognitive impairment

Not all forgetful patients have true dementia (e.g. Alzheimer's disease).

Sometimes, especially after being depressed or after a major life event, a false dementia can develop. It is called (Pseudodementia) or (depressive dementia).

Suspect **Pseudodementia** in an elderly who has been **depressed** e.g. due to a **major life event** + they are **aware of their symptoms** e.g. memory loss.

In **True dementia**, patients **do not** have insight to their illness; they are usually brought to the hospital by others.

On the other hand, in **pseudodementia**, the patients themselves notice their symptoms (e.g. memory loss, social withdrawal, not careful, impaired concentration) and usually **present to the hospital on their own**.

A final difference, Alzheimer's (True dementia) is a chronic slowly progressive disease while **Pseudodementia** has a **relatively abrupt onset**.

Example (1),

A 70 YO \bigcirc presents to the hospital complaining of forgetfulness for 3 months. Her husband died 4 months ago. For the last 3 months, she has been forgetful, more socially withdrawn. She is late in paying her bills. She contacts her children less frequently than usual. She had Depression 20 years ago.

The likely $Dx \rightarrow Pseudodementia$ (Depressive Dementia).

- √ She presents to the hospital on her own (has insight to her symptoms).
- **▼** Relatively abrupt onset.
- √ The symptoms begin after a major life event (the death of her husband).

Important, although this may seem as normal grief response (< 6 months after a major life event), the features are more towards pseudodementia.

Have a look on the features of Normal Grief Response

Normal Grief Response			
Stage -1: Hours to days	Denial Disbelief Numbness		
Stage -2 : Weeks to 6 months	Sadness, weeping, waves of grief. Somatic symptoms of anxiety Poor sleep, Guilt, Blame of others, Illusions, hallucinations Preoccupation with memories of the deceased Social withdrawal		
Stage-3: Weeks to months	Symptoms resolve Social activities resumed Memories of good times www.Plab1keys.com		

Grief Denial & Isolation \rightarrow Anger \rightarrow Depression \rightarrow Bargaining \rightarrow Acceptance

Example (2),

A 70 YO \bigcirc was found by the police wandering the streets in the middle of the night and brought to the ED. His neighbour told the doctors that he has been living alone after his wife died 1 year ago. They also said that he has increased forgetfulness, getting lost easily when outside home, talking to himself in the past nine months

The likely $Dx \rightarrow Dementia$

Not Pseudodementia!

- Brought to the hospital by others (does not have insight to his illness).
- The onset is chronic (9 months-1 year).

Key 59

- Seizure lasting > 5 minutes → Status epilepticus.
- Management in community → Buccal midazolam or Rectal diazepam
- Management in hospital or if there is IV access → IV Lorazepam
- Any of which is repeated twice before giving the second step drug which is
- → IV Phenytoin.
- If still ongoing, the third step would be → refer to ICU.

Key 60

- OLD man + GDU (Gait abnormality/ Dementia -behaviour changes-/ Urine urgency ± incontinence) = Wet, Wobbly, Wacky Grandpa. (if CT is done, it would show enlarged ventricles)
- → NPH (Normal Pressure Hydrocephalus).

- OLD + GDU (Gait abnormality/ Dementia -behaviour changes-/ Urine urgency ± incontinence) = Wet, Wobbly, Wacky Grandpa. But, with Hx of HTN, Smoking, TIAs, MRI shows multiple lacunar old infarcts
- → (Vascular Dementia).
- Disinhibition (Inappropriate actions and sayings e.g. inappropriate sexual comments/ urinating on sofa) → Frontal Lobe
- Struggling with word choices → Temporal Lobe
- → Frontotemporal dementia (Pick's Disease)

(Even if there is Hx of HTN and smoking. In Vascular dementia, there would usually be Hx of cardiac or vascular events e.g. TIAs)

Key If a patient with delirium has become aggressive/ agitated/ distressed and he
 61 has no Dementia, Alzheimer's, or Parkinson's → Acute psychosis.

We could consider Haloperidol or Olanzapine as a short-term therapy if non-pharmacological (de-escalation) techniques have failed.

Key CT scan showing a lesion in a cerebral hemisphere in a patient who hasseizure.

The cause of seizure is \rightarrow **Space-occupying lesion** (That is seen on the CT).

(Note: **MRI** is better than CT for soft tissues. However, a CT is also useful).

• **Space-occupying lesions** (eg, Abscess, Tumour, Hematoma) can cause seizures.

V In **Huntington's disease** → jerky, random uncontrolled movements (Chorea) that start to appear between the age of 35-45 YO. (Autosomal dominant).

 \forall In **Epilepsy** \Rightarrow There has to be 2 unprovoked seizures with > 24 hours apart.

Inattention or **neglect** in one side + **Seizure** + **Neurological deficit** (e.g. hyperreflexia of a limb/ weakness of a limb

→ Think of **Space-occupying lesion** such as cerebral tumor or cerebral abscess (if there is fever, headache, source of infection)

Key Remember, 63

- **♦** Lucid intervals
- → Epidural (Extradural) hemorrhage
- → Middle Meningeal Artery.
- ♦ Hx of fall, elderly, progressive symptoms ± Hx of Alcoholism/ Anticoagulation
- → Chronic Subdural Hematoma
- → Bridging (cerebral veins).

Key 64 A 35 YO \bigcirc presents with headache on the back of the head, and pain on flexing her neck for several days. The pain is aggravated by moving the neck. O/E, there is a limited neck movement.

The likely $Dx \rightarrow Cervical Spondylosis$.

- ♦ It cannot be meningitis (no fever, photophobia, vomiting, not acute...etc).
- ♦ It cannot be subarachnoid or subdural hemorrhage → intact level of consciousness and mentation, not acute nor progressive.
- ◆ Cervical Spondylosis = limited and painful neck movement + occipital headaches. (Related to Activity). It can develop later into radiculopathies.

Key 65

Cerebral Abscess VS Cerebral Tumor VS Viral Encephalitis

Cerebral Abscess

- = Features of meningitis (Mainly Severe Persistent Headache) with fever
- + Obvious nearby **source of infection** (eg, ear pain with discharge)
- + Neurological deficit such as limb weakness (as it is a space-occupying lesion)

CT scan \rightarrow Ring-enhancing lesion.

- MRI is better than CT in visualising soft tissue.
- However, CT is also useful and is requested if MRI is contraindicated.

Example (1),

A 44 YO \bigcirc presents with headache, neck stiffness and vomiting. The headache is severe and persistent and on the left side. She also has left ear pain with discharge and weakness of right hand and leg. Her temperature is 38.6 C.

The likely $Dx \rightarrow$ Cerebral Abscess

Key 66

Example (2),

A 66 YO man has just had a seizure and he is now conscious and oriented. O/E, inattention on the left side is noticed with hyperreflexia of the arm.

The likely $Dx \rightarrow \frac{\textbf{Cerebral Tumour}}{\textbf{Cerebral Tumour}}$ (space-occupying lesion)

- ◆ Inattention or neglect in one side + Seizure + Neurological deficit (e.g. hyperreflexia of a limb/ weakness of a limb → Think of Space-occupying lesion such as cerebral tumor or cerebral abscess (if there is fever, headache, source of infection)
- **♦ Inattention** or neglect is a feature of parietal lobe lesion.

Example (3),

31 YO presents with headache, confusion and photophobia that are gradually getting worse over the past week. Temperature is 38.2. There is No Neck stiffness. Negative Kernig's sign. LP is done and **no** organism was found on CSF culture. CT head is unremarkable.

The likely $Dx \rightarrow Viral Encephalitis$. (A Diagnosis of Exclusion).

- ◆ Neck stiffness, Kernig's and Brudzinski Signs are commonly seen in bacterial meningitis. (they are not present here, so likely not bacterial meningitis).
- ♦ If **cerebral abscess**, the stem would have mentioned a *nearby source of infection*. Furthermore, the CT here is unremarkable while in Cerebral Abscess it would show *ring-enhancing lesion*.
- ♦ If **TB meningitis**: *Mycobacterium Tuberculosis would have appeared on culture* (a bit easily detected). Also, Acid fast bacilli sometimes show on CSF smear.

Key A child + Stares blankly into space (daydreaming) + After that, resuming their
 activities, they are tired and not doing well ± upturning of eyes/ or eyelids fluttering.

→ Absence seizure

Rx might be required if they are recurring a lot

→ Sodium Valproate.

Key 68

♦ In intracranial tumour

- → Give high does dexamethasone "initially" to shrink the mass and edema and therefore alleviate the headache and the other symptoms.
- ♦ Note, if GCS is ≤ 8 , we give "Mannitol" as it has a very rapid action. Otherwise, we start with corticosteroids (high dose dexamethasone is preferred). Remember: GCS ≤ 8 is an indication for intubation.
- ♦ In Intracranial Hemorrhage with **Very Low GCS** and Neurological deficit (e.g. **Unequal Pupils**)
- → Urgent Craniotomy

Key 69

Sensory Loss Responsible Nerve Roots:

3 in the thigh \ 2 in the shin \ 1 in the foot

- Groin and pelvic Girdle → L1
- Anterior thigh → L2
- Inner (Medial) thigh and distal anterior thigh \rightarrow L3
- Inner (medial) shin → L4

- Outer (Lateral) shin and Dorsum of the foot → L5
- Lateral Foot → S1

Scenario 1:

A man develops severe low back pain shooting down his right leg after lifting heavy objects. His Ankle and Knee reflexes are intact. He has reduced sensory stimulus over the dorsum of the right foot.

The likely nerve root affected \rightarrow L5

Scenario 2:

A patient with DM presents for routine check-up. His reflexes and motor functions are normal. However, there is a deficit in fine touch sensation on the medial aspect of his lower right leg.

The likely dermatome to be affected \rightarrow L4

(inner shin = Medial side of a leg = L4)

Key 70

- ullet In intracranial hemorrhage (e.g. Hx of fall) ullet CT scan is the most appropriate.
- In Stroke (ischemic) \rightarrow MRI is most appropriate (however, we initially order CT).

Key 71

Amaurosis Fugax:

Painless, Temporary, Unilateral, and Recurrent loss of vision that lasts from a few seconds to a few minutes due to embolism (transient occlusion) of the **Central retinal artery**.

- Usually lasts for 5 − 15 minutes and resolves within < 24 hours.
- It is a transient ischemic attack (TIA) → CT head to R/O hemorrhagic stroke.
- Risk Factors:

Atherosclerosis (Bruit on neck) | Hypertension | Giant Cell Arteritis (GCA)

- A patient may describe it as "A black Curtin Coming Down his vision".
- The embolus in Amaurosis Fugax comes from atherosclerotic Internal carotid artery while in Transient Ischemic Attack (TIA), the emboli of the cerebral hemispheres come from the heart.
- First → CT scan head to rule out hemorrhage.
- TIA long-term Rx → Aspirin for 2 weeks followed by clopidogrel 75mg for life.

Key 72

■ Combined oral contraceptive pills (COP) are Absolutely Contraindicated in patients with Migraine with aura. (↑ risk of cerebrovascular accident)

- The **best** contraception in patients with migraine with aura is
- → Copper Intrauterine Device.

Migraine with Aura:

The headache starts before the end of the aura or within an hour after the end of the aura. (Important \lor)

Aura examples:

◆ Visual signs and symptoms → Fortified spectra (Flashes of light), Homonymous hemianopia, Scotoma (blind spots)

♦ Others → Unilateral paraesthesia "Numbness, Tingling", Speech or language difficulty "Dysphasia", Muscle weakness

Key 73

- DVT + Fullness (Deafness, Vertigo, Tinnitus) + Pressure or Fullness in one ear
- → Meniere's disease (MRI is normal). Give buccal or IM (Prochlorperazine)
- DVT + Cranial Nerve Palsy (e.g. Facial palsy, Loss of Corneal reflex, Loss of facial sensation) ± fullness
- → Acoustic Neuroma (Vestibular Schwannoma)

(Do MRI of the cerebellopontine angle).

Tinnitus = ringing in ear.

Key 74

Neurofibromatosis (NF) (Autosomal Dominant)

- NF type 1 presents more with skin lesions. (e.g. Café-au-lait spots "brown macules" / Axillary or Groin Freckles / iris hamartomas / scoliosis / association with Pheochromocytoma).
- NF type 2 presents more with CNS tumours. (e.g. Bilateral acoustic neuroma / multiple intracranial schwannomas, meningiomas)

Left facial pain that's sharp, shooting, electric-like, radiates to left check and Key 75 ↑ with chewing, lasts for seconds then repetitive throughout the day. → Trigeminal Neuralgia. The most appropriate $Rx \rightarrow Carbamazepine = (Anticonvulsant)$. The hint that is usually given in the exam for Multiple Sclerosis is optic neuritis Key 76 (Pale ± Swollen optic disc, Blurry vision) + Remitting and Relapsing + Other Neurological manifestations. The Definitive Diagnostic test → MRI brain and spinal cord → Demyelination, Lesions disseminated in time and place. The Rx of acute MS → IV Methylprednisolone ■ Painless muscle weakness that worsens with exercise, **Tires easily**, Speech Key 77 fades, Difficulty climbing stairs and reaching for items on shelves, Difficulty chewing and swallowing, Diplopia, Normal reflexes ± FHx of an autoimmune disease (e.g. Thyroid disease) (Positive autoimmune panel) The likely Ds → Myasthenia Gravis \rightarrow To confirm Dx

→ Serum skeletal muscle nicotine acetylcholine receptor antibody

- The same presentation as Myasthenia gravis **but** with the following **differences**:
- V The reflexes are absent and elicited after exercise
- √ Strength/ power of the weakened muscles after repeated test
- → Lambert-Eaton Syndrome (Associated with tumours)

Important:

- ♦ Myasthenia Gravis association → Thyrotoxicosis.
- ◆ Lambert-Eaton Syndrome association → Tumours (e.g. small cell lung cancer)

In Lambert-Eaton Syndrome, the muscle function IMPROVES with use.

Key Stroke → Sudden focal neurological loss due to vascular origin lasting for > 24
 78 hrs.

 \blacksquare TIAS \rightarrow Sudden focal neurological loss due to vascular origin lasting for < 24 hrs.

Example,

A 66 YO smoker and hypertensive patient presents with a sudden onset weakness of the right arm with dysphasia that resolved within 24 hours.

◆ The likely Dx → Transient Ischemic Attack (TIA). (Resolved within 24 hours)

◆ The best <u>next</u> modality → Carotid Doppler Scanning

Carotid duplex should be done within 2 weeks of admission to check for carotid artery stenosis to assess for the need of carotid endarterectomy.

When to perform Carotid endarterectomy?

 \lor If internal carotid artery stenosis is ≥ 50% in \circlearrowleft (Men)

 \forall If internal carotid artery **stenosis** is ≥ **70%** in \bigcirc (**Women**)

Key 79

Remember,

- Amyotrophic lateral sclerosis (ALS) "a Motor Neuron Disease"
- → Motor weakness (progressive dysphagia and complete paralysis later)
- + Normal autoimmune panel
- + Deep reflexes are (1), **positive** for upper neuron disease.
- Myasthenia Gravis → Similar to (ALS) but with Positive autoimmune panel + Normal deep reflexes. (usually eyes are affected; Diplopia, Drooping eyelids)
- Lambert-Eaton Syndrome (Associated with tumours e.g. Lung cancer)

The same presentation as Myasthenia gravis **but** with the following **differences**:

V The reflexes are absent and elicited after exercise.

√ ↑ Strength/ power of the weakened muscles after repeated test. ♦ In Intracranial Hemorrhage (e.g. after fall from height) with Very Low GCS Key 80 and Neurological deficit (e.g. Unequal Pupils) → Urgent Craniotomy. This is done after Intubation and referral to neurosurgical team to evacuate the hematoma. Key Remember, 81 Whenever you see $GCS \le 8 \rightarrow \text{immediately think of } Intubation (or: Inform$ the anaesthetist). Remember, Key 82 **Horner's syndrome** → Unilateral *Ptosis, Miosis, Anhidrosis* Due to → Compression of the Ipsilateral Sympathetic Chain. (e.g. in Pancoast tumor) High blood glucose (e.g. Due to infusion of 5% Dextrose) Key 83 + Infusion of hypotonic solution (e.g. 0.45% NaCl) for prolonged period \rightarrow Hyponatremia (\downarrow Na+) → Cerebral Oedema "Osmotic type" → Confusion, drowsiness

◆ Avoid rapid correction of hypernatremia especially with hypotonic solution → "it can cause cerebral edema" due to abnormal pressure gradient issues that you do not need to understand deeply for PLAB 1".

When Sodium is low → Your brain will blow!

Whenever there is hyperglycemia, there is osmotic flow of water from ICF to ECF.

Key 84

Remember,

If limb ataxia → Cerebellar Lobe is affected "ipsilateral side"

If tRuncal ataxia → Cerebellar VeRmis is affected. (The midline vermis of cerebellum)

Key 85

Remember,

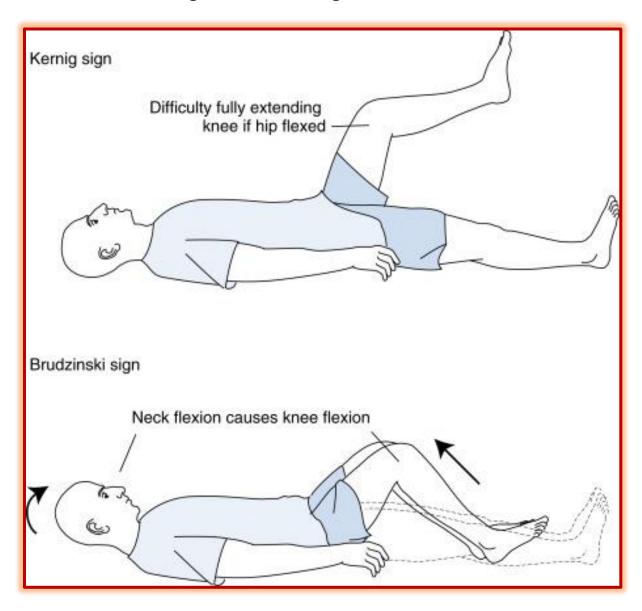
Tongue biting Urinary incontinence unconscious Post-ictal Confusion are characteristics for \rightarrow Epileptic Seizures.

Another episode is required to be given a diagnosis of Epilepsy.

 \forall In **Epilepsy** \Rightarrow There has to be **2 unprovoked seizures with > 24 hours apart**.

Key Neck stiffness, Kernig's and Brudzinski Signs are commonly seen in bacterial
 meningitis. (Others: Severe headache, photophobia, high fever)

If Rash → think of meningococcal meningitis



For the Investigation:

 \forall If without rash, pick \rightarrow Lumbar puncture (LP) = CSF Analysis.

√ If with Rash, pick → Blood culture (Meningococcal Septicemia – Neisseria Meningitidis)

Key 87

DVLA "Driver and Vehicle Licensing Agency"

- Q1) An elderly woman with Alzheimer's disease continues to drive and refuses to inform any authority. What should you do?
- → Inform DVLA "Driver and Vehicle Licensing Agency"
- It is the responsibility of the patient to inform DVLA.
- If they refuse to do so, it is now your responsibility as a doctor to inform DVLA.
- Q2) A man has a <u>single</u> TIA "Transient Ischemic Attack" and he is now fit for discharge. What should you advise him about car driving?
- → Stop car driving for at least 1 month
- → Stop lorry or Bus driving for 1 year

When to inform DVLA?

- If group A driver (Car) and has **MULTIPLE TIAs** within a short period.
- If group B driver (Lorry or Bus) and has a SINGLE TIA.

Q3) For the first time, a Lorry driver has an <u>epileptic seizure</u>. What should be done?

→ He needs to inform DVLA and should not drive until certain criteria are met.

In seizures (Not TIAs):

- A Car driver can drive again after 1 year of being seizure-free.
- A Lorry driver can drive again after 5-years of being seizure free.

Key For the exam,

88

If you have to choose between Subarachnoid hemorrhage and Acute subdural hemorrhage, Pick "Acute subdural hemorrhage" if Hx of trauma followed by Rapid deterioration.

SAH is usually with "the worst headache" "very intense over the back-occiput-", "Thunderclap headache" Again, Trauma is more associated with Acute subdural hemorrhage then with Subarachnoid hemorrhage.

Key 89 Stiff- freezed- posture, Axial rigidity → Falling Backwards, shuffling and freezing gait, restricted downward gaze + Others

→ Supranuclear palsy.

Key 90 Diabetic Amyotrophy (Proximal diabetic Neuropathy)

- A complication of poorly controlled DM. (nerve disorder)
- Severe thigh or leg pain → followed by Proximal Muscular Wasting e.g. shoulders, thighs (quadriceps) muscles, pelvic girdle (Proximal leg).
- Good diabetic control is a must.
- Usually resolves within months.

N.B. Vasculitis neuropathy presents similarly but with ↑↑ ESR.

Key 91 31 YO presents with headache, confusion and photophobia that are gradually getting worse over the past week. Temperature is 38.2. There is

No Neck stiffness. Negative Kernig's sign. LP is done and no organism was found on CSF culture. CT head is unremarkable.

The likely $Dx \rightarrow Viral Encephalitis$. (A Diagnosis of Exclusion).

- ◆ Neck stiffness, Kernig's and Brudzinski Signs are commonly seen in bacterial meningitis. (they are not present here, so not bacterial meningitis).
- ♦ If **cerebral abscess**, the stem would have mentioned a *nearby source of infection*. Furthermore, the CT here is unremarkable while in Cerebral Abscess it would show *ring-enhancing lesion*.
- ♦ If **TB meningitis**: *Mycobacterium Tuberculosis would have appeared on culture* (a bit easily detected). Also, Acid fast bacilli sometimes show on CSF smear.

Key While driving his car, a 33 YO man developed seizure, Loss of consciousness and with tongue biting.

The likely $Dx \rightarrow \frac{\text{epileptic seizure}}{}$

	Epilepsy	Non-Epileptic Attack Disorder (NEAD)
	Genetic Factors	Hx of child abuse (either sexual or physical)
Duration of the episode	Less than 2 minutes	More than 2 minutes
Pelvic Movement	X	V
Asynchronous movements	X	V
Eye	Open	Closed
If eye closed	Easy to open manually	Difficult to open manually
Drooling of saliva	٧	X
Tongue biting	V	X
Self-injury during attack	٧	X
Urine incontinence		X
Post-ictal confusion	V	X
Post-ictal EEG	Slow	Normal

Key A 44 YO ♀ presented with blurred vision and intermittent clumsiness for 3
 months. Reflexes are brisk in her arm and the optic disc is pale.

The best modality to confirm $Dx \rightarrow MRI brain$ "likely Multiple Sclerosis"

The best drug in acute phase → Methylprednisolone

Stiffness + Weakness + Optic Neuritis + Recurrence/ intermittent → MS

Multiple sclerosis \rightarrow MRI brain \rightarrow Methylprednisolone

Key A patient presents with <u>Parkinson's</u> features + Visual <u>hallucinations</u> + **94** Dementia

→ Lewy Body Dementia

Dx -> MRI brain followed by Dopamine transporter uptake imaging

REMEMBER:

- ♠ Parkinson's features + Visual hallucinations + Dementia
- → Lewy Body Dementia.
- ♠ Parkinson's features + Urinary incontinence + Postural hypotension
- → Shy-Drager Syndrome.

- ♠ Old man + Gait abnormality + Dementia + Urinary incontinence [GDU]
- → Normal Pressure Hydrocephalus.
- ◆ Old + Making sexual or inappropriate comments (*Disinhibition*) + Loss of social interest (*disengagement*) + Acting inappropriately or impulsively + Personality and behaviour changes ± Over-eating
- → Frontotemporal dementia (Pick's disease).

Key An alcoholic man wants to quit. He wants a medication to help reduce the withdrawal symptoms

→ Chlordiazepoxide

(The question does not ask about a medication that acts as a **deterrent**; they need a medication to **Reduce Withdrawal Symptoms**)

✓ An alcoholic wants a medication to serve as a <u>Deterrent</u> when he takes alcohol "Abstinence" \rightarrow <u>Disulfiram</u>.

A child + Stares blankly into space (daydreaming) + After that, resuming their activities, they are tired and not doing well ± upturning of eyes/ eyelids fluttering.

→ Absence seizure

Rx might be required if they are recurring a lot

→ Sodium Valproate.

Key On the 2nd day in hospital, a patient has become restless, agitated and sees spiders on the bed.

→ Delirium Tremens.

(He is likely a **chronic alcohol addict**, and being in a hospital and not drinking for 2 days may lead to firstly alcohol withdrawal symptoms. If **hallucinations**/ **tremors**/ **delusions** develop, the case is called **delirium tremens**, and the patient should be given **IV Lorazepam**).

Key A patient with lung cancer presents with ptosis and miosis.

The likely cause → Compression of the Ipsilateral Sympathetic Chain

Horner's syndrome → Unilateral *Ptosis, Miosis, Anhidrosis*

Due to → Compression of the Ipsilateral of Sympathetic Chain.

(e.g. in Pancoast tumor)

 Pancoast Tumour → A tumour of the Apex of the Lung (present at the top end of either the left or the right lung). It typically spreads to the nearby tissues such as the Ribs, the Vertebrae, unilateral compression of sympathetic chain, causing Horner's syndrome. Most Pancoast tumours are Non-small cell lung cancer.

So, in an elderly who is **smoker** and present with **chest pain** (often pleuritic) + signs of Horner's (e.g. unilateral **miosis**, **ptosis**),

suspect → Lung cancer (particularly: Pancoast tumor).

Key
 A 56 YO old woman, Severe headache, Chronic kidney disease,
 Hypertension. Her brother died at age of 40 due to cerebrovascular problem.

The likely $Dx \rightarrow Subarachnoid hemorrhage$.

- ♦ Polycystic Kidney Disease → Berry Aneurysm → Subarachnoid Hemorrhage
- ◆ Polycystic Kidney disease is an autosomal dominant (strong FHx as seen here).

lacktriangle Commonly asked association \rightarrow berry aneurysm \rightarrow subarachnoid hemorrhage.

Polycystic Kidney Disease (ADPKD):

(Hypertension and repeated kidney stones) or (HTN + CKDs with FHx)

√ Association: Berry Aneurysm → Subarachnoid hemorrhage (Important √)

√ Another Association → SIADH → Hyponatremia. (Important √)

Key In any patient on Warfarin, the most important symptom that he needs to urgently report is

→ HEADACHE

(This is because people on warfarin are liable to subdural hematoma which presents with headache and other features)

- In any patient on **Bisphosphonates**, the most important symptom that needs to be urgently reported is
- → Severe, sudden Heartburn or Chest pain (either is correct)

Key A 36 YO presents to the ED with a Severe headache with vomiting for 1 day.

The headache started when he was lifting weights in a gym. He has

photophobia and neck stiffness and GCS of 12/15. A CT head is ordered and it shows:



His BP is normal with mild tachycardia. Which drug is useful in this case?

[Aspirin or: Clopidogrel or: Sumatriptan or: Nimodipine]

√ Firstly, this is a case of **Subarachnoid hemorrhage (SAH)**.

 \lor The hyperintense areas on the CT \rightarrow blood in the subarachnoid basal cisterns.

√ In SAH, cerebral vasospasm can occur 4-12 days later and it is severe. ■ To diminish this anticipated cerebral vasospasm, → we give Calcium Antagonist (e.g. Nimodipine). ■ The strongest **genetic** risk factor for **Alzheimer's** disease (AD) Key 102 → APOE ε4 gene Acute alcohol withdrawal symptoms (sweating, agitations, tremors, altered Key 103 mentation) → Chlordiazepoxide "First" + then give Thiamine (Vit. B1) If with "seizure" or "Hallucination" [i.e. Delirium Tremens] → IV Lorazepam. Or Diazepam "If IV Lorazepam is not in the options) Wernicke's encephalopathy (CAS: Confusion, Ataxia, Squint: ophthalmoplegia, Nystagmus, diplopia), → IV Vitamin B1 (Thiamine) (IV Pabrinex) or (High potency Vitamin B Complex). ✓ An alcoholic wants a medication to serve as a <u>Deterrent</u> when he takes alcohol "Abstinence" \rightarrow **Disulfiram**.

- √ An alcoholic wants a medication to help <u>reduce withdrawal symptoms</u>
- → Chlordiazepoxide.

V An alcoholic wants a medication to reduce his Craving for alcohol → Acamprosate.

Example 1,

3 days post-hernioplasty, a 55 YO patient has become agitated, aggressive, confused and developed auditory hallucination.

Hb (normal), MCV 112 (high), Gamma-GT (high), ALP (normal).

The most appropriate management \rightarrow Lorazepam.

√ Although this stem does not mention a Hx of chronic alcoholism, we can spot this from the raised MCV and GGT.

Note, in a stem with post-operative patient develops confusion, aggressiveness, suspect → Chronic alcohol consumption.

✓ Since the patient has not been drinking alcohol for 3 days, he developed **Delirium tremens** (**Hallucination** is the clincher).

√ If the symptoms developed in 6-24 hours after the surgery and there was no hallucination or seizures, it would only be "Acute Withdrawal Symptoms" such as sweating, tachycardia, anxiety, tremors. We would give

→ **Chlordiazepoxide** in this case.

√ If CAS (Confusion, Ataxia, Squint: ophthalmoplegia/ Diplopia), this would be
Wernicke's encephalopathy. We would give

→ Thiamine (Vit B1)

Example 2

On the fourth day post-operative day, a woman has become confused and she sees spiders on her bed.

The likely $Dx \rightarrow$ Delirium tremens

Give → Lorazepam

(She is likely a **chronic alcoholic** and now develops **hallucinations** -seeing spiders- which indicate **delirium tremens** for which **IV Lorazepam** is used)

Key ■ An alcoholic man wants to quit. He wans a medication to help reduce the 104 withdrawal symptoms → Chlordiazepoxide (they did not ask about a medication to act as a deterrent; he needs a medication to Reduce Withdrawal Symptoms) Key ☐ On the fourth day post-operative day, a woman has become confused and she sees spiders on her bed. 105 The likely $Dx \rightarrow$ **Delirium tremens** (She is likely a chronic alcoholic and now develops hallucinations -seeing spiders- which indicate delirium tremens for which IV Lorazepam is used) Cauda Equina Syndrome → Perianal/ groin numbness (Saddle Paraesthesia) Key **Inability to initiate voiding "urination"** Back pain. 106 ■ Next step → Urgent MRI

Key ■ An elderly man presents with bruises. He is disoriented and confused.

He takes warfarin.

107

→ Subdural hematoma.

Key A Heavy smoker and alcoholic admitted for a fractured humerus. On the 3rd day of admission, he developed confusion, hyperreflexia, hypertonia and nystagmus.

What's the most important medication to give?

→ IV Thiamine.

Wernicke's encephalopathy (CAS: Confusion, Ataxia, Squint: ophthalmoplegia, Nystagmus, diplopia), may present 12-24 hours after stopping alcohol.

Give \rightarrow IV Vitamin B1 = (Thiamine) = (IV Pabrinex)

or (High potency Vitamin B Complex).

A man with upper respiratory infection. He then developed confusion, anxiety and aggression. He is having lethargy intermittently. He says people are watching him and there are cameras watching him. What's is the most likely diagnosis?

- A) Alzheimer's disease
- B) **Delirium**
- C) Lewy body dementia

- D) schizophrenia
- E) Mania
- **ACUTE** onset (hours to days) of mood and behavioural changes + Hallucinations (mainly visual)
- → Delirium
- Elderly, UTI, or Resp. infections, developed confusion, fluctuating level of consciousness and disoriented to time and place/ delusions/ hallucinations.
- → Delirium
- Urinary Tract Infections (UTIs) are very common cause of Delirium in Elderly.
- Key
 25-year-old woman with progressive weakness and loss of sensation that
 started from the lower limbs. Currently she is finding it difficult to swallow.
 She had malaise and bouts of diarrhoea 2 weeks ago. What mechanism explains the symptoms she is having?
 - a) Demyelination of CNS
 - b) Demyelination of PNS (of nerve fibres)
 - c) Spinal cord compression
 - d) Antibodies to muscular end plate

She had GIT infection. Now developed Guillain barre syndrome.

- √ The mechanism of Guillain barre syndrome
- → Autoimmune degeneration of myelin sheets of the peripheral neurons.
- \forall For Dx \rightarrow Nerve conduction study.
- V Ascending weakness (usually begins in LL) + Loss/reduced tendon reflex
- **± Hx of GIT or Respiratory infection**
- → Guillain Barre Syndrome

Left facial weakness with inability to close the left eye + ptosis. What is the therapeutic approach?

- a) Steroids (Prednisolone)
- b) Aspirin
- c) No treatment indicated
- d) Acyclovir

Facial (Bell's Palsy) = 7th nerve palsy.

V Rx (Important) → Prednisolone (Corticosteroids) = Steroids. Burning Pain. Type 1 Diabetes Mellitus with retinopathy and nephropathy. Key Which drug to give for his neuropathic pain? (No amitriptyline in options) 112 A. Naproxen B. topiramate C. **Duloxetine** D. Steroids In Diabetic Neuropathy: Neuropathic pain can present in any form of the following: (Burning), (Tingling), (Numbness), (Itching), (Paraesthesia), (Shooting/ **Stabbing Example**, a diabetic patient with ankle ulcer with agonising Burning Pain. Rx? -> Amitriptyline (1st line) or Gabapentin or Duloxetine or Pregabalin. Away Goes D neuropathic Pain

(+) Good glycemic control

Key A 68yr old woman with severe intermittent right sided facial pain that is sharp and Stabbing. Most appropriate management

- A. anticonvulsants
- **B. NSAIDs**
- C. steroids
- D. SSRIs
- E. Tricyclic Antidepressants

Trigeminal Neuralgia 5th CN.

Unilateral Electric shock-like pain/ sharp, shooting stabbing pain in one side of the face (unilateral) which is sudden, episodic and lasts for a few seconds to minutes with recurrence. It might start in the jaw angle and radiate to the temporal region or forehead.

MM. blab TreAsion

The pain is worsened on chewing, movement or touch.

Rx → Medication is tried first, then surgery

√ Medications examples → Carbamazepine (First line) (Important √)

Others → lamotrigine, phenytoin, gabapentin.

Sometimes, the answer will be (Anticonvulsants). Carbamazepine is an anticonvulsant.

 \vee Surgery \rightarrow microvascular decompression.

Note, in trigeminal neuralgia, corneal reflexes are usually **intact**.

- 59-year-old man with lower limb and shoulder weakness and pain when trying to stand from a chair. He is a known diabetic on metformin. Muscular atrophy is notes in shoulder and lower limbs. His HbA1C is 67, ESR-22, CK-mildly elevated. What is the most appropriate diagnosis?
 - A) Diabetic Amyotrophy
 - B) Diabetic peripheral neuropathy
 - C) Polymyalgia rheumatica
 - D) Polymyositis

Diabetic Amyotrophy (Proximal diabetic Neuropathy)

• A complication of poorly controlled DM. (nerve disorder)

- Severe thigh or leg pain → followed by Proximal Muscular Wasting e.g. shoulders, thighs (quadriceps) muscles, pelvic girdle (Proximal leg).
- Good diabetic control is a must.
- Usually resolves within months.
- 76-year-old man with problems finding words and short-term memory.

 Tendency to cry but no persistent low mood. The patient has a History of hypertension for 12 years. What is the most likely Diagnosis?
 - A. Frontal lobe dementia
 - B. Vascular dementia
 - C. Alzheimer's dementia
 - D. Lewy body dementia

Dementia + Hx of HTN, Smoking, TIAs, MRI shows multiple lacunar old infarcts

Think of → Vascular Dementia.

Key A man was admitted to a hospital. After a few days, he developed ataxia, ophthalmoplegia. What medication can be used for his condition?

A. A. Chlordiazepoxide

- B. Thiamine
- C. Lorazepam

te

E. Disulfiram

Chronic alcoholic + CAS (Confusion/ Ataxia/ Squint "Nystagmus, Ophthalmoplegia)

- → Wernicke's encephalopathy (Vitamin B1 -Thiamine- deficiency).
- © Chronic alcoholic ± CAS + Amnesia (memory loss) + Confabulation (Making up stories) → Korsakoff's psychosis
- Old man with 20 years history of Parkinson's, now with dementia, cognitive dysfunction. Now has visual and tactile hallucinations for 6 months with increased cognitive impairment. What is the likely Dx?
 - A. Alzheimer
 - b. Vascular Dementia
 - c. Lewy body Dementia
 - d. Parkinson's associated dementia.
 - E. Picks dementia

A patient presents with <u>Parkinson's</u> features + Visual <u>hallucinations</u> + Dementia

→ Lewy Body Dementia.

REMEMBER:

- ♠ Parkinson's features + Visual hallucinations + Dementia
- → Lewy Body Dementia.
- ♠ Parkinson's features + Urinary incontinence + Postural hypotension
- → Shy-Drager Syndrome.
- ◆ Old man + Gait abnormality + Dementia + Urinary incontinence [GDU]
- → Normal Pressure Hydrocephalus.
 - B. ♠ Old + Making sexual or inappropriate comments (*Disinhibition*) + Loss of social interest (*disengagement*) + Acting inappropriately or impulsively + Personality and behaviour changes ± Over-eating
- → Frontotemporal dementia (Pick's disease).

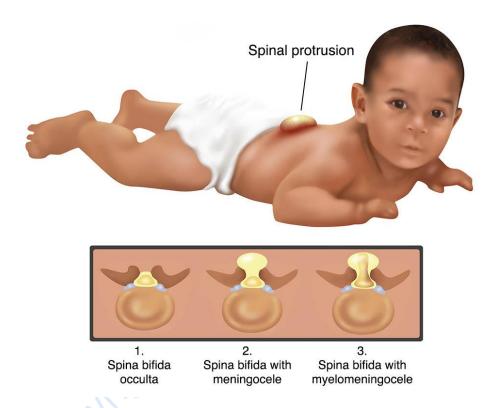
Key 118

Remember, the Rx of:

- **V** Bell's palsy → high doses prednisolone for 10 days.
- √ Ramsay Hunt syndrome → antiviral (acyclovir).
- **V** Trigeminal neuralgia → Carbamazepine (anticonvulsant).

Spina bifida

(Important points)



- Spina bifida is a birth defect in which there is incomplete closing of the spine and the membranes around the spinal cord during early development in pregnancy.
- There are three main types: spina bifida occulta, meningocele and myelomeningocele.
- The most common location is the lower back, but in rare cases it may be in the middle back or neck.
- Occulta has no or only mild signs, which may include a hairy patch, dimple, dark spot or swelling on the back at the site of the gap in the spine.

- Meningocele typically causes mild problems, with a sac of fluid present at the gap in the spine.
- Myelomeningocele, also known as open spina bifida, is the most severe form. Problems associated with this form include poor ability to walk, impaired bladder or bowel control, accumulation of fluid in the brain (hydrocephalus), a tethered spinal cord and latex allergy. Learning problems are relatively uncommon.
- Most cases of spina bifida can be prevented if the mother gets enough folate before and during pregnancy.
- Adding folic acid to flour has been found to be effective for most women.
- Open spina bifida can be surgically closed before or after birth.
- A shunt may be needed in those with hydrocephalus, and a tethered spinal cord may be surgically repaired.
- Devices to help with movement such as crutches or wheelchairs may be useful.
- Urinary catheterization may also be needed.
- Prophylactic antibiotics are useful to prevent recurrent UTIs.
- Intermittent self-catheterisation is useful in cases of healed UTI with persistence high bladder pressure and urinary retention.
- If there is still <u>ongoing UTI</u>, <u>urinary incontinence pad</u> is preferred over intermittent self-catheterisation.

Folic acid Dosage in Pregnancy

- \blacksquare The usual dose \rightarrow 0.4 mg (400 ug) a day for 12 weeks of pregnancy.
- **5** mg a day for 12 weeks of pregnancy if any of the following:
- √ DM.
- √ BMI > 30.
- √ A pregnant woman taking **antiepileptics**.
- √ FHx of NTD (Neural Tube Defect).
- √ Previous pregnancy with NTD.
- **5** mg for the entire length of pregnancy if:
- V Thalassemia or thalassemia trait.
- √ Sickle Cell Disease (SCD).

Key 120 V Having physical disability (e.g. due to stroke which has led to one arm paralysis) can prevent patients from doing daily activities such as brushing their teeth, getting dressed, preparing food ...etc.

V Occupational therapists can help such patients to perform these daily activities by teaching them special one-handed techniques.

Key 121

Important Notes about meningitis organisms:

- Gram +ve diplococci → Streptococcus pneumoniae.
- Gram -ve diplococci → Neisseria Meningitidis.
- Gram +ve cocci in grape-like clusters, Coagulase and Catalase positive
- → Staphylococcus aureus.
- Gram +ve bacilli → Listeria monocytogenes.
- Gram -ve coccobacilli → H. influenza.

Also,

 \forall Turbid (or purulent) CSF (+) No rash \rightarrow think streptococcus pneumoniae.

 \forall Turbid (or purulent) CSF (+) there is rash \rightarrow think Neisseria Meningitidis.

Key 122

Symmetrical ascending weakness preceded by an infection (e.g. URTI, Gastroenteritis)

→ Guillain barre syndrome

To diagnose \rightarrow Nerve conduction study.

Key 123	Suspecting SAH and CT, MRI are inconclusive? Do LP.		
Key 124			
	A) Inform DVLA. B) Ask him to inform DVLA. C) Look through his records to find if he has had recent seizure.		
	People with epileptic seizure while awake that causes loss of consciousness can still drive their cars provided that:		
	• 1 year of no seizure.		
• > 6 months of no seizure if they have changed their medicati			
	So, it is appropriate to find out if he has suffered epileptic attacks during the last year first before advising him to inform DVLA.		
	co64		
Key	Transient Ischemic Attack: site of lesion based on the affected organ:		
125	 Leg → Anterior communicating artery (ACA). 		
	 Face and Arms → Middle cerebral artery (MCA). 		
	 Vision and language → Posterior cerebral artery (PCA) "asked recently √" 		

Key
 V If a non-pregnant woman is taking Sodium Valproate to control her seizures,
 and no alternative is available, advise here to use reliable contraception as
 sodium valproate is very teratogenic.

(She needs to avoid pregnancy while on this drug).

Key 127 A 55 YO man with a Hx of hypertension presents with 1 week of headache and blurry vision of both eyes. On examination, his left eye is displaced outward and downward. What is the most appropriate investigation?

→ MRI of the head.

- This is likely a case of a space-occupying lesion.
- It can cause oculomotor palsy as seen in the stem.
- MRI is better than CT, however, both are good options.

Gerstmann Syndrome

- The characteristic features are inability to designate or name the different fingers of the two hands (finger agnosia),
- confusion of the right and left sides of the body,
- and inability to calculate (dyscalculia)
- □ to write (dysgraphia).
- One or more of these manifestations may be associated with word-blindness (alexia) and homonymous hemianopia or a lower quadrantanopia,
- lesion is in the inferior parietal lobuleparticularly the angular gyrus or subjacent white matter of the left hemisphere

In a recent exam, it was asked about the anatomical site that is involved?

→ Parietal lobe

129

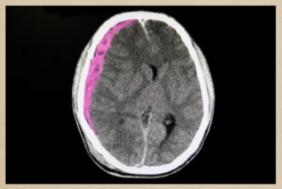
Key In a recent exam, a head CT showing biconvex hematoma is given and was asked about the diagnosis. The answer was epidural "extradural" hematoma. See the comparisons below:

- Epidural = Lemon
- Subdural = Banana

Remember these key facts:

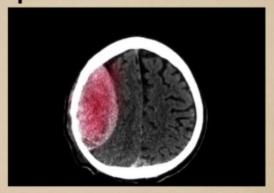
- **Epidural Hematoma** Abnormal collection of blood between skull –nd dura
- √ Convex, lens-shaped
- √ Injury to middle meningeal artery
- √ Blunt trauma, especially to pterion
- √ Lucid interval
- **Subdural Hematoma** Abnormal collection of blood between dura a–d arachnoid
- √ Concave, crescent-shaped
- √ Injury to bridging veins
- √ Accelerating-decelerating injury
- √ Elderly, chronic alcoholism

Subdural Hematoma



- Concave/Crescent-Shaped
- Bridging Veins
- Elderly, Alcoholics

Epidural Hematoma



- Convex/Lens-Shaped
- Middle Meningeal Artery
- "Lucid Interval"

- 130 An eldery man is brought by his son to the A & E. This morning, he started to suffer from confusion, seeing people walking in his room, and inability to remember what happened yesterday. He had eye check 2 weeks ago and it was normal. He looks drowsy. What is the next investigation?
 - → serum sodium.
 - This man is having acute delerium "started today morning".
 - In the A&E, check electrolyte first as hyponatremia can cause acute delerium.
 - Other causes for acute delerium include infections, drug abuse, alcohol withdrawal, acute MI, head injuries, DM, thyrotoxicosis.

• CT of the head would be needed to exclude stroke, space-occupying lesions. However, in "acute" delerium, blood and urine tests are to be ivestigated first such as electrolytes, infections, hypoglycemia.

Key 131 An eldery man is brought by his son to a GP surgery clinic. He has been suffering from confusion, seeing people walking in his room, seeing flowers in the room that he know they are not real. These complaints have been there for 3 months now. He does not have auditory hallucinations. The confusion is <u>occasional</u> in the morning. He had eye check 2 weeks ago and it was normal. What is the single most appropriate investigation?

- → MRI scan of the head.
- This man is having Lewy body dementia, for which "MRI brain" is the investigation of choice.
- In the previous key, the visual hallucinationa and confusion were acute "started this morning", which means (Delerium). Here, it is chronic over 3 months, which means (Dementia).

Lewy Body Dementia criteria:

- ◆ Dementia (the usual presenting feature) with memory loss and ↓ problem solving ability.
- ◆ **Fluctuating** levels of awareness and attention.

- ♦ Mild Parkinsonism (e.g. Rigidity, Tremors, Ataxia, Falls) "not always a feature".
- ♦ Visual Hallucination (e.g. humans, animals) and illusions. (Important √).
- → Request MRI brain (of choice in Lewy body dementia).

◆ Transverse Myelitis

→ Weakness (e.g. in legs), Spastic Quadriparesis or Spastic Paraparesis
 Urinary urgency/ retention
 Stiffness
 ↑ tone and brisk reflexes.

Key 133

- Unilateral throbbing headache, associated with nausea, vomiting, tinnitus, visual disturbance
- → Migraine with aura.
- Bilateral throbbing headache, associated with nausea, vomiting, tinnitus, visual disturbance + occurs daily + worse in the morning + improves with standing + obesity is a risk factor
- → Idiopathic intracranial hypertension (IIH).

Key 134

A 67 YO man presents with sudden onset blurred vision, speech difficulties and vertigo started a few hours ago. There is no unilateral limb weakness.

O/E, he has horizontal nystagmus and dysarthria. A few hours later, these symptoms improved but he is still having dysarthria. His GCS is 15 and his CT head is normal.

 \lor The likely $Dx \rightarrow Posterior circulation stroke.$

 \lor The most appropriate action \rightarrow MRI of head.

- The cerebellum is partly supplied by the posterior inferior cerebellar artery.
- Ischemia to cerebellum → vertigo, ataxia, nystagmus.
- The occipital lobes are supplied by the posterior cerebral artery.
- Ischemia to the occipital lobes → visual disturbances.
- MRI of the head is the investigation of choice for cerebellar infarction.
- Generally, in acute stroke → CT without contrast (but it is of a <u>limited value</u> in posterior circulation stroke).
- Other features of Posterior Circulation Stroke
- → Nausea, vomiting, dysarthria, gaze-evoked jerk nystagmus, unilateral limb weakness and numbness, gait ataxia.

Dysdiadochokinesia = inability to perform rapid alternating muscle movements.

135

Key | A female on combined oral contraceptive pills (COCPs) presents with features of hemiplegic migraine with aura (weakness of an arm, tingling in face, increased sensitivity to light and sound, unilateral throbbing headache) after a few hours, her symptoms resolve. CT is normal.

→ Advise her to stop COCPs as it increases the risk of ischemic stroke.

Key 136

In ischemic stroke, what is the medication that would reduce the chance of a long-term brain injury if given within 4.5 hours of the symptom's onset?

→ Alteplase (thrombolytic therapy).

Note that antiplatelets (clopidogrel, aspirin) and statins (atorvastatin) are given to prevent a further ischemic stroke. However, they do not reduce the chance of a long-term brain injury, alteplase does.

Key 137

Parkinson's disease dementia

√ Occurs more than a year after diagnosis of Parkinson's disease.

√ Its distinct features include:

• limbs rigidity.

- fluctuations in lucidity.
- visual hallucinations.

Q1) A man with Parkinson's disease on co-careldopa (Sinemet) for 2 years now started to have low moods (depression), muscle stiffness and rigidity, and frequent fluctuations of lucidity. He also has cognitive difficulties with memory and executive functions' problems.

The likely cause of his symptoms \rightarrow Parkinson's disease dementia.

"2 years have passed; it is hard to be a side effect of co-careldopa. Also, side effects of co-careldopa do not include lucidity fluctuations or rigidity. They may include dyskinesia, hallucinations, focal tremors.

Q2) A man with Parkinson's disease on co-careldopa (Sinemet) for around 3 weeks. Now, he started to have low moods (depression), involuntary erratic muscle movements (dyskinesia), and focal tremors.

The likely cause of his symptoms \rightarrow Side effects of co-careldopa.

√ Side effects of co-careldopa includes dyskinesia (most importantly), depression, focal tremors ± hallucination.

V Also, the duration between the start if the medication and the beginning of the symptoms is short. This indicates the more likelihood of the drug being causing these new symptoms.

A 35 YO woman has severe headache for 4 hours, and the headache is worsening gradually. This started with visual disturbance (large black dots in her vision = scotoma), tingling of her right arm that has spread to her mouth, and the difficulty to find the right word to use in a sentence. There is no motor weakness or vertigo.

The most likely $Dx \rightarrow Migraine$.

This is likely a case of migraine with <u>aura</u>.

If this was a case of cerebral infarction; "stroke or TIA", she would have slurred speech (dysarthria) instead of the difficulty to find a right word (which is called word salad).

A quick comparison between Migraine with aura and Stroke/TIA:

Migraine with aura	Stroke/ TIA
Gradual onset	Sudden onset
Mainly visual symptoms e.g., scotoma "large black dots"/ zigzags "fortification spectra".	May have a temporary loss of vision.
Sensory: tingling/ numbness affecting one arm, spreading to or around mouth.	May have unilateral numbness.
Word salad: difficulty to find the right words.	Dysarthria: slurred speech.

-	± Dysphagia: difficulty swallowing
± motor weakness, vertigo.	± motor weakness (e.g., hemiparesis), vertigo.

The safest anti-epileptic medications in pregnancy

139

→ Lamotrigine, followed by Carbamazepine.

Key 140

Functional Weakness of LL = non-organic cause

Hoover's sign used in the diagnosis of functional weakness of the lower limb.

+ve Hoover's sign:

When the patient is lying supine, flexion of his "weak" hip against resistance will **NOT** be followed by extension of the other "normal" hip.

Think → Functional weakness.

Whereas in **normal** people or those with **organic cause** of lower limb weakness such as multiple sclerosis, flexion of the weak hip against resistance **will be followed** by extension of the other normal hip **(-ve Hoover's sign)** = normal/organic cause.

Any **suspected case of epilepsy** "seizing for 1st time" presenting to A&E with features suggesting epilepsy eg, tongue biting, post-ictal confusion, ≤2 min.

→ refer to first-fit clinic.

There would be neurologists who will take detailed history and give a management plan.

Key 142 A 36 YO woman presents to a neurology clinic complaining of episodes of temporary weakness of left arm and numbness on her left face. This is followed by headache on left side of her head. The episodes are recurrent around twice a month, each can last for up to 24 hours. Her blood pressure is normal. What is the most suitable PROPHYLACTIC medication?

- √ The likely Dx here is Hemiplegic migraine with aura.
- ✓ Note that the question asks about a treatment used as **prophylaxis** (to reduce the frequency of the symptoms). In this case:
- **V** The first-line prophylactic medication is → Beta blockers (e.g., Propranolol).
- ✓ If the question asks about the first-line management in **acute attack**, the answer would be (oral sumatriptan: if >17 YO) or (Nasal sumatriptan: if 12-17 YO) with or without NSAIDs/paracetamol.

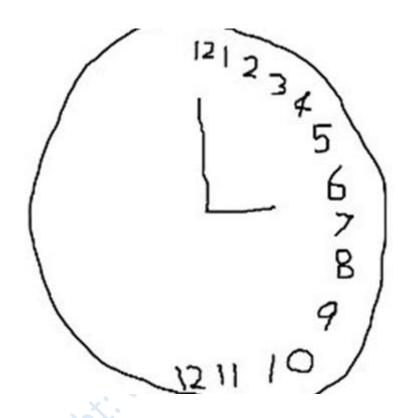
Note that propranolol is for prophylaxis (not beneficial during the attacks).

Important note:

If this woman is on COCP \rightarrow Advise her to switch to Progesterone-only pills.

(COCP is contraindicated in patients who are suffering from migraine with aura as it increases the risk of ischemic stroke).

Key After a stroke, a patient has become unaware of objects or people on one side. He is unaware of his problem. He was asked to draw a clock, and this was the result:



- \blacksquare The likely Dx \rightarrow Hemineglect Syndrome. (Neglect Syndromes).
- \blacksquare The likely affected structure \rightarrow Parietal lobe "important".

Also → **Spatial** memory difficulties (eg, cannot navigate around her local area)

 \rightarrow Parietal lobe. (Spatial = Parietal). (Spatial; from space).

Key 144 ■ Parkinsonism [+] Dementia [+] Visual Hallucinations ± Delusions

→ Lewy Body Dementia.

Investigations → **MRI brain** "to R/O other causes"

Followed by → **SPECT** (Single-photon emission computes tomography)

That is also known as (DatSCAN) ie, Dopamine transporter uptake imaging.

Key 145

<u>Alzheimer's</u> disease patients with <u>LBBB</u> (M shaped or notched R wave in V6). ± bradycardia ± Prolonged PR intervals.

Give \rightarrow **Memantine**.

• Acetylcholinesterase inhibitors (which are first-line in Alzheimer's) such as rivastigmine and donepezil are not suitable in heart conditions as they may lead to pronounced bradycardia and heart block "prolonged PR intervals". In such cases, memantine is an alternative.

Key 146

Notes on Parkinson's Treatment

- Co-careldopa can cause → nausea and vomiting as side effects.
- How to avoid nausea and vomiting in Parkinson's disease patients who take co-careldopa?
- **∨** By taking co-careldopa with food.

If no improvement:

- **√** Use antiemetics.
- What is the antiemetic of choice in patients with Parkinson's disease?
- → Cyclizine. V Others: domperidone.

Although ondansetron can be use in Parkinson; however; not ideal for elderly!

Note that:

V [May Cause Parkinson] ie, Metoclopramide, Cinnarizine and Prochlorperazine are <u>contraindicated</u> in Parkinson's disease patients as they may worsen symptoms.

 \lor Other medications to <u>avoid</u> in <u>Parkinson's</u> \rightarrow Haloperidol and Olanzapine.

Key 147

Central Post-Stroke Pain [CPSP]

- Central post-stroke pain (CPSP) is a <u>neuropathic pain</u> (shooting pain, stabbing pain, electrical pain, or hypersensitivity, or numbness or loss of sensation) that can occur days, months or even years after a cerebrovascular accident (Stroke).
- This syndrome is characterised by pain and sensory abnormalities in the body parts that correspond to the brain territory that has been injured by the cerebrovascular lesion.
- Management → Gabapentin, or Pregabalin or Amitriptyline (Neuropathic).

Other Notes:

- Baclofen is a skeletal muscle relaxant that can be used in <u>muscle spasms</u> that might occur in multiple sclerosis, spinal cord injury or after stroke.
- Carbamazepine is the drug of choice in trigeminal neuralgia.

V Never use Haloperidol or Metoclopramide in patients with Parkinson's:

• The rapid tranquilizer (in **delirium**, **psychosis**, **agitated** patients) used in **Parkinson's** patients → **Lorazepam**. "Can be given IV or IM in urgent cases".

Key 149

- Palliative team should <u>not</u> try to prolong their palliative patients' lives as this would prolong their suffering. Also, they should <u>not</u> try to shorten their lives or lead to their death (Euthanasia is prohibited and illegal in the UK).
- The aim of palliative care register is to make patients comfortable at the last hours of their lives.
- **Example**: An elderly man with lung cancer and metastasis who is bed-bound and unable to do his daily activities had fallen on his head and developed intracranial hemorrhage.
- \rightarrow Give anxiety treatment (sedation) eg, \rightarrow Midazolam.

(Midazolam can be given subcutaneously).

Key 150

Sudden-onset of **nystagmus**, **gait ataxia**, **vertigo** -**<u>Dizziness</u>**-, homonymous **hemianopia**, **dysdiadochokinesia**:

 \lor The likely $Dx \rightarrow$ Posterior Circulation Stroke.

- Generally, in acute stroke → CT without contrast (but it is of a <u>limited value</u> in posterior circulation stroke).
- Other features of Posterior Circulation Stroke

→ Nausea, vomiting, dysarthria, gaze-evoked jerk nystagmus, unilateral limb weakness and numbness, gait ataxia, Dizziness (vertigo).

Dysdiadochokinesia = inability to perform rapid alternating muscle movements.

Key 151

An Alzheimer's/ Dementia Patient Requires Cognitive Assessment. Which Cognitive Assessment Tool to Use?

- If the patient presents to a **1ry care** eg, **Emergency department** or **GP**.
- → 6-item Cognitive Impairment Test (6-CIT), which is quick and fast (6 questions only) and more appropriate in the Emergency (ER) and GP settings.
- If the patient presents to a 2ry care eg, Memory clinic/ or Neurology clinic (not in the ER or primary care as GP), eg, Alzheimer's patient presents to a memory or neurology clinic and needs an assessment for his cognitive impairment:
- **V** Mini-mental State Examination (MMSE) or
- **V** Montreal Cognitive Assessment (MoCA) or
- **V** Addenbrooke's Cognitive Examination (ACE). ACE is more beneficial in complex cases that require comprehensive assessment (that involves changes in personality + Language + Behaviour ...etc).
- Note: to assess Alcohol Consumption and identify individuals who may have harmful drinking patterns \rightarrow AUDIT-C.

An old lady says inappropriate sexual words to her young grandson. Key 152 This is called \rightarrow Disinhibition. Seen in → Fronto-temporal dementia (Pick's disease). The likely affected anatomical structure → Orbito-frontal lobe. V Remember these medications: Key 153 Cerebral Toxoplasmosis (multiple homogenous ring enhancements on image) → Pyrimethamine + Sulfadiazine. Cryptococcal meningitis (cryptococcal antigen is detected on CSF) → Amphotericin + Flucytosine. **Quick Notes on Brain Tumor** Key 154 • Headache (worse in the morning). • Papilledema (optic disc swelling + venous engorgement). → MRI scan of the brain (preferred over CT scan in brain tumors). If MRI is contraindicated \rightarrow CT scan.

What is the Occluded Artery? (A quick reminder)

- Sudden complete loss of vision in the right eye
- The likely occluded artery → **Left** internal carotid artery. (Contralateral).
- Right-sided hemiplegia + Right homonymous hemianopia
- The likely occluded artery → **Left** middle cerebral artery. (Contralateral).

Key 156

Important Question (Previously Asked):

What is expected to be seen on the MRI of a chronic alcoholic who suffers from Amnesia (memory impairment)? In other words, Wernicke's Korsakoff syndrome can be seen in chronic alcohol consumers. One of its features is Amnesia (memory impairment). Which brain structures that are responsible for this amnesia?

→ Mamillary bodies and thalamic regions. (Responsible for memory).

(MRI would show → Mamillary bodies atrophy).

Important Notes on Hepatic Encephalopathy:

- The underlying **mechanism** of hepatic encephalopathy is:
- → The buildup of <u>ammonia</u> in the blood. (Normal liver usually removes ammonia but if liver fails like in hepatic encephalopathy, ammonia builds up in the blood).
- The patient would be severely **confused** (due to ammonia in blood).
- The first line management is → Lactulose.
- V Lactulose decreases ammonia production by bacteria.
- V Lactulose decreases intestinal absorption of ammonia.

Key 158

Any patient with a history of chronic **headache** and then develops a <u>new onset</u> strabismus should have **CT scan** of the head to rule out **intracranial mass**.

History of **Headache** + New onset **strabismus** \rightarrow **CT scan** of head.

Strabismus — also known as hypertropia and crossed eyes — is misalignment of the eyes, causing one eye to deviate inward (esotropia) toward the nose, or outward (exotropia), while the other eye remains focused. A convergent deviation of eye.

2 Important Neurology Scenarios, Pick the Difference:

(1)

- Stiff- freezed- posture (decreased speed), Loss of balance, Axial rigidity → (Falling Backwards), shuffling and freezing gait, restricted downward gaze + Others (eg, minor tremors and neck rigidity).
- → Progressive Supranuclear Palsy.

(2)

Tremors of both hands, losing balance, (<u>falling forward</u>), slow gait and difficulty to stop, leaning forward when walking, slow and monotonous speech, tense muscles (rigidity), **No** downward gaze restriction.

- → Idiopathic Parkinson's Disease
- Supranuclear palsy → Falling backward + Downward gaze restriction.
- Parkinson's → Falling forward + No downward gaze restriction.

Key 160

Multiple System Atrophy (MSA)

- It is a rare neurodegenerative disease. It is considered one of the Parkinson's plus syndromes (or: Atypical Parkinsonism). It is similar to idiopathic Parkinson disease (IPD) in some features but distinct and different in other features.
- Similar to IPD in: tremors, rigidity, bradykinesia (parkinsonism features).

<u>Multiple System Atrophy (MSA)</u> is Different from Idiopathic Parkinson's Disease (IPD) in the following Points:

- √ Typically, MSA has an earlier onset (it starts in the age between 50-60 years).
- √ MSA parkinsonism (tremors, rigidity) is often bilateral.
- √ MSA had significant autonomic dysfunction (recurrent urinary tract infections, postural hypotension eg, light-headed on standing).
- √ The "bent-over" posture is more severe and pronounced in MSA than in IPD.
- V MSA commonly has stridor and high-pitch noise during sleep.
- √ MSA often has cerebellar dysfunction (eg, dysarthria).
- √ MSA tends to progress more rapidly than IPD.

Key 161

Stepwise cognitive impairment (progressive cognitive impairment in an old patient with a history of hypertension ± smoking)

Think → multi-infarcts dementia.

- Multi-infarcts dementia is a type of vascular dementia caused by small infarcts (strokes) in different areas of the brain.
- The symptoms may include difficulties in memory, language, thinking behaviour and may also include problems in speech, balance and coordination.
- The keyword is (step-wise cognitive decline).

Management of Psychosis in Parkinson's Patients:

- Acute psychosis in Parkinson's patient → Lorazepam. "Rapid tranquilizer".
- If the psychosis is ongoing on for several days → Clozapine / Quetiapine.

Key 163 Myalgic Encephalomyelitis (ME) = Chronic Fatigue Syndrome (CFS)

Features of ME/CFS:

• **Fatigue** → The hallmark symptom, severe disabling fatigue that is not relieved by rest and it impacts daily activities.

In depression, the hallmark complaint is low mood. Here, the hall mark is fatigue.

- **Post-exertional malaise** → Fatigue is worsened by physical or mental activity even if minimal activity, and not significantly relieved by rest.
- > 50% of the cases start with \rightarrow a flu-like illness.
- Cognitive impairment → often difficulties in memory, concentration ...etc.
- Pain and discomfort (not uncommon) → widespread pain eg, muscle and joint pain, pain on touch, sore throat, headaches.
- Other symptoms → sleep disturbance, mood disturbance, symptoms ↑ with standing.

Rapid eye movement (REM) Sleep Behaviour Disorder (RBD)

- It is a parasomnia characterized by dream-enactment behaviours that emerge during a loss of REM sleep atonia. It is commonly associated with **Parkinson's**.
- V Dream enactment ranges in severity from benign hand gestures to violent thrashing, punching, and kicking (patients act out their dreams).
- **V** Second- line or adjunctive $Rx \rightarrow Melatonin$.

Mnemonic $\Theta \rightarrow Clon$ azepam = make clone of sleepwalkers.

Key 165

Quick Note:

People with <u>Down Syndrome</u> have a markedly increased risk of developing <u>Alzheimer's dementia</u> at a younger age compared to the general population.

Quick Question:

A 48-year-old man has diabetes, hypertension, down syndrome, epilepsy (on antiepileptic medications). He has recently become more forgetful, and his cognitive function had been declining. What is the major cause for his dementia? (DM, Hypertension, Down syndrome, antiepileptic drugs)?

→ Down syndrome.

Key 166

Making **sexual** or **inappropriate** comments (**Disinhibition**):

→ Orbitofrontal lobe problem.

	Disinhibition manifests as inappropriate social and sexual behaviour, poor judgment, and impulsivity.
Key 167	Pseudodementia → Cognitive deficit (eg, forgetful) + depression.
	It is just like an extreme form of grief. Ie, the cognitive symptoms are a manifestation of depression rather than rue dementia.
Key	Headache due to cerebral metastasis
168	1^{st} line \rightarrow Dexamethasone.
	Let's
Key 169	A common complication that may arise after a sudden cessation of dopaminergic therapy eg, co-careldopa (a medication for Parkinson's disease)
	is → Akinesia (loss of voluntary muscle movement).
Key	is → Akinesia (loss of voluntary muscle movement). This is why co-careldopa is usually continued even in the patients who will undergo surgery (in the perioperative period) for both elective and emergency surgeries. Poorly controlled DM (nerve disorder) + Severe thigh or leg pain → followed
	is → Akinesia (loss of voluntary muscle movement). This is why co-careldopa is usually continued even in the patients who will undergo surgery (in the perioperative period) for both elective and emergency surgeries. Poorly controlled DM (nerve disorder) + Severe thigh or leg pain → followed by Proximal Muscular Wasting e.g. shoulders, thighs (quadriceps) muscles
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Key 171

The artery that supplies the majority of the occipital lobe, including the visual cortex is the → Posterior cerebral artery.

Example: Bilateral right hemianopsia (sudden transient painless loss of vision in the right halves of both eyes) → Posterior cerebral artery (PCA).

Key 172

Scenario: Acute Onset of Agitation and Confusion

A 40-year-old man presents to the Emergency Department with an acute onset of confusion, disorientation, and agitation. His family reports that his symptoms began suddenly 8 hours ago. He is afebrile, and his physical examination shows no signs of meningeal irritation or focal neurological deficits. A CT scan of the brain reveals no abnormalities. Which is the most likely diagnosis?

- A) Intracerebral hemorrhage.
- B) Streptococcal meningitis.
- C) Cerebral toxoplasmosis.
- D) Viral encephalitis.
- E) Tuberculosis meningitis.

Answer → **D) Viral encephalitis** is the most likely diagnosis.

Explanation: The patient's presentation of acute onset confusion, disorientation, and agitation with a normal CT scan and absence of meningeal irritation or focal neurological deficits suggests an infectious or inflammatory process affecting the brain parenchyma.

Why Viral Encephalitis?

- Viral encephalitis can present with acute onset of confusion, agitation, and other mental status changes.
- The absence of fever does not rule out encephalitis, as some cases may present without fever initially.
- The normal CT scan does not exclude encephalitis, as imaging may be normal in the early stages.
- This diagnosis is often one of exclusion, as other causes of acute mental status changes are ruled out.

Why Not the Other Options?

A) Intracerebral hemorrhage:

 Typically presents with focal neurological deficits, headache, and can be detected on a CT scan. The normal CT scan and absence of focal deficits make this less likely.

B) Streptococcal meningitis:

 Meningitis usually presents with fever, neck stiffness, and altered mental status. The absence of meningeal signs and a normal CT scan make bacterial meningitis less likely.

C) Cerebral toxoplasmosis:

• Commonly seen in immunocompromised patients, especially those with HIV/AIDS. It often presents with focal neurological deficits and ringenhancing lesions on CT/MRI, which are not seen in this case.

E) Tuberculosis meningitis:

 Typically presents with subacute or chronic symptoms, including headache, fever, and cranial nerve palsies. The abrupt onset and absence of systemic symptoms make this less likely. **Summary**: In patients presenting with acute onset confusion, disorientation, and agitation without focal deficits or meningeal signs, and with a normal CT scan, viral encephalitis is a likely diagnosis. This is often an answer of exclusion, made after ruling out other potential causes. Early recognition and treatment are critical to prevent complications and improve outcomes. Further evaluation, including lumbar puncture and cerebrospinal fluid analysis, would be necessary to confirm the diagnosis and identify the causative virus.

Viral Encephalitis (Summarised Topic)

- **©** Common Viruses that can cause Viral Encephalitis in the UK:
- Herpes Simplex Virus (HSV): Predominantly HSV-1.
- Varicella Zoster Virus (VZV).
- Enteroviruses: Includes coxsackievirus and echovirus.

Clinical Presentation:

- Fever.
- Headache.
- Altered Mental Status: Confusion, agitation.
- Seizures.
- Focal Neurological Deficits.
- Sensory and Motor Disturbances.

Patients may also show symptoms of a preceding viral infection, such as respiratory or gastrointestinal illness. Compared to viral meningitis, viral encephalitis presents more with signs of CNS involvement like confusion and seizures.

Diagnosis:

- Lumbar Puncture for CSF Analysis: Essential for diagnosis; findings similar to viral meningitis.
- CT Scans: Often normal; used to rule out other causes like stroke, hemorrhage.
- MRI Scans: More useful for detecting inflammation and edema.

■ Management of Viral Encephalitis (UK Guidelines):

- 1. Hospital Admission: Admit for monitoring and supportive care.
- 2. Intravenous Acyclovir: (14-21 days).
- 3. **Supportive Care**: Ensure hydration, pain management, and fever control.
- 4. Empirical Antibiotics: Used until bacterial meningitis is excluded.
- 5. Intensive Care: Transfer to ICU if severe symptoms are present.

Key Points:

- Early Acyclovir is crucial.
- Supportive care and monitoring are essential.

Key 173

Scenario: Managing Acute Agitation in Parkinson's Disease

A 74-year-old woman with a 6-year history of Parkinson's disease, who is currently an inpatient, has become increasingly agitated and verbally aggressive. The nursing staff report that her agitation intensifies in the evening, and she has been experiencing significant sleep disturbances for the

past week. Her current medications include levodopa and a monoamine oxidase inhibitor. Which of the following is the most appropriate management for her acute agitation?

- A) Baclofen.
- B) Propranolol.
- C) Clonidine.
- D) Midazolam.
- E) Chlorpromazine.

Answer → **D) Midazolam** is the most appropriate management for her acute agitation.

Explanation: The patient's agitation and verbal aggression, especially in the context of Parkinson's disease, need to be managed carefully to avoid exacerbating her Parkinsonian symptoms.

Why Midazolam?

- Midazolam is a benzodiazepine that is effective for the rapid control of acute agitation and can be administered intramuscularly or intravenously.
- It provides quick sedation, reducing agitation and allowing for better management of the patient.
- Benzodiazepines are generally well-tolerated in patients with Parkinson's disease and do not worsen Parkinsonian symptoms.

Why Not the Other Options?

A) Baclofen:

• A muscle relaxant used for spasticity, not effective for managing acute agitation or aggression in this context.

B) Propranolol:

 A beta-blocker used primarily for hypertension and anxiety-related tremors. It is not suitable for acute agitation and can worsen symptoms in Parkinson's patients due to its central nervous system effects.

C) Clonidine:

 An alpha-2 agonist used for hypertension and certain withdrawal symptoms. It is not first-line for acute agitation and can cause hypotension and sedation.

E) Chlorpromazine:

An <u>antipsychotic</u> that can <u>worsen Parkinson's symptoms</u> due to its
dopamine antagonist effects. It is **not recommended** for patients with
Parkinson's disease.

Summary: Midazolam is the best choice for managing acute agitation in a patient with Parkinson's disease, providing rapid sedation without worsening Parkinsonian symptoms. Other medications like baclofen, propranolol, clonidine, and chlorpromazine are either ineffective for this purpose or potentially harmful in this patient population.

Key 174

Scenario: Managing Restless Legs Syndrome

A 60-year-old man presents to the clinic complaining of uncomfortable sensations in his legs, particularly in the evenings, which disrupt his sleep. He

describes these sensations as a deep-seated urge to move his legs, sometimes accompanied by tingling and crawling feelings. These symptoms temporarily relieve when he walks around or stretches his legs but return upon resting. On examination, there are no abnormal neurological findings. His recent full blood count and renal function tests are within normal limits. Which of the following is the most appropriate first-line pharmacological treatment for this patient's condition?

- A) Sertraline.
- B) Clonazepam.
- C) Ropinirole.
- D) Amitriptyline.
- E) Indapamide.

Answer → C) Ropinirole is the most appropriate first-line pharmacological treatment for this patient's condition.

Explanation: The patient's symptoms are characteristic of restless legs syndrome (RLS), a condition marked by an uncomfortable urge to move the legs, usually worsening in the evening and at rest, and relieved by movement.

Why Ropinirole?

- **Ropinirole** is a dopamine agonist, which is considered first-line treatment for RLS. Other dopamine agonists: Ropinirole, Pramipexole, Rotigotine.
- It helps to alleviate the symptoms by modulating dopamine pathways,
 which are thought to be involved in the pathophysiology of RLS.

Why Not the Other Options?

A) Sertraline:

 An SSRI used for depression and anxiety, which can sometimes exacerbate RLS symptoms.

B) Clonazepam:

 A benzodiazepine that can help with sleep disturbances but is not the firstline treatment for RLS and can cause dependency.

D) Amitriptyline:

 A tricyclic antidepressant that can exacerbate RLS symptoms and is not a first-line treatment.

E) Indapamide:

 A thiazide-like diuretic used for hypertension, which is not relevant to the treatment of RLS.

Restless Legs Syndrome (RLS) - Overview

Symptoms:

√ Urge to Move Legs:

 Persistent need to move the legs, especially during periods of rest or inactivity eg, feeling the need to move legs while sitting or lying down.

V Uncomfortable Sensations:

• Tingling, crawling, or creeping feelings in the legs.

• Example: Describing the sensation as "ants crawling" on the skin.

V Worsening at Evening/Night:

• Symptoms intensify in the evening or night, disrupting sleep.

√ Temporary Relief with Movement:

- · Symptoms are temporarily relieved by walking or stretching.
- Example: Needing to walk around the room to reduce discomfort.

√ Sleep Disturbances:

- Difficulty falling or staying asleep due to leg discomfort.
- Example: Waking up frequently at night to move the legs.

■ Diagnosis:

- Based on clinical presentation.
- Rule out secondary causes (e.g., iron deficiency, renal insufficiency).
- **First-Line Treatment** → **Dopamine agonists**: Ropinirole, Pramipexole.

■ Other Management Options:

- Gabapentin, Pregabalin, Levodopa (for intermittent use).
- Address underlying conditions (e.g., iron deficiency).
- Lifestyle modifications: Regular exercise, sleep hygiene.

Key 175

Contraception Option in Epileptic Women on Sodium Valproate

A 34-year-old woman with a history of epilepsy is advised by her neurologist to begin treatment with sodium valproate. During her consultation, she seeks advice on contraception, mentioning that she might want to become pregnant within the next few years. Which of the following contraceptive methods would be the most appropriate for her to start before initiating sodium valproate?

- A) Progesterone-only pill (POP).
- B) Levonorgestrel-releasing intrauterine system (Mirena coil).
- C) Copper intrauterine device (IUD).
- D) Combined oral contraceptive pill (COCP).
- E) Medroxyprogesterone acetate injection (Depo).

Answer → B) Levonorgestrel-releasing intrauterine system (Mirena coil).

Explanation:

- High Teratogenic Risk: Sodium valproate has a high risk of causing birth defects, so it is crucial to ensure highly effective contraception in women of childbearing age, even if she desires a short-term contraception!
- LARC (Long-acting Reversible Contraceptive): The Levonorgestrelreleasing intrauterine system (Mirena coil) is a long-acting reversible
 contraceptive, which is one of the most effective forms of contraception,
 with a typical-use failure rate of less than 1%. This is essential for women
 on sodium valproate, as preventing unplanned pregnancy is critical.
- Long-term, Reliable Protection: The Mirena coil offers protection for up to 5 years and requires no daily action, reducing the chance of user error and ensuring consistent contraception during sodium valproate treatment.
- Copper IUD (Option C) is another highly effective option, but it lacks the hormonal benefits of the Mirena coil, such as cycle regulation and reducing menstrual bleeding.
- Progesterone-only pill (POP) (Option A), COCP (Option D), and Depo
 injections (Option E) are less reliable due to higher typical-use failure rates
 and the need for strict adherence or frequent dosing, which could lead to
 user error.

Mirena's Hormonal Benefits: In addition to being highly effective, the
 Mirena coil provides hormonal benefits that can help regulate menstrual cycles and reduce heavy menstrual bleeding, which may be an added advantage for some women.

Therefore, **B)** Levonorgestrel-releasing intrauterine system (Mirena coil) is the most appropriate contraceptive method due to its reliability, long-term protection, and minimal maintenance.

Additional Notes:

If she is planning to conceive in the near future, her neurologist should consider alternative anti-epileptic drugs (AEDs) that are safer during pregnancy, such as lamotrigine or levetiracetam, both of which are considered to have lower teratogenic risks compared to sodium valproate.

Key 176

Driving Restrictions in Epilepsy: Ensuring Compliance and Safety

A 42-year-old man presents to his GP for a routine follow-up after being diagnosed with epilepsy two years ago. He reports that his last seizure occurred three months ago. He is stable on his current anti-epileptic medication regimen and tolerating it well. At his last visit, the GP advised him to stop driving.

However, he admits during this visit that he has continued driving, stating that he feels well and believes he can drive safely. What is the most appropriate next step in managing this patient?

- A) Advise the patient to drive with caution.
- B) Contact the hospital to discuss the case further.
- C) Refer the patient for a neurological review.
- D) Inform the patient that you will report him to the DVLA.
- E) Encourage the patient to stop driving until seizure-free for 12 months.

Answer → D) Inform the patient that you will report him to the DVLA.

Explanation:

• First Step - Advise and Encourage: Initially, when a patient with epilepsy experiences a seizure, the GP should advise the patient to stop driving and encourage them to refrain from driving until they have been seizure-free for at least 12 months. This is consistent with UK law, which requires a 12-month seizure-free period before resuming driving. This step was already taken during the patient's previous GP visit.

- Continued Driving: Despite this advice, the patient has continued to drive, which places both the patient and the public at risk. Although the patient feels well, the fact that they have not been seizure-free for 12 months makes driving unsafe and illegal.
- **GP's Duty to Inform the DVLA**: As the patient has not followed the advice and continues to drive, it is now the GP's legal and ethical responsibility to report this to the **DVLA** (Driver and Vehicle Licensing Agency). This is necessary to protect public safety. The GP should inform the patient that if they do not report their condition to the DVLA, the GP is required to do so to prevent any harm.
- Encouraging the patient to stop driving (Option E) is still part of the
 management, but since the patient admits to continuing driving despite
 previous advice, informing the DVLA becomes a crucial step to ensure public
 safety.
- Other options such as advising caution (Option A) or referring for a neurological review (Option C) do not address the immediate risk of the patient driving. Contacting the hospital (Option B) would not be the next step either, as the priority is to prevent the patient from continuing to drive.

In summary:

As a first step, GPs advise and encourage patients to stop driving until they are seizure-free for at least 12 months. However, if the patient continues to drive against this advice, it is the GP's duty to report this to the **DVLA** to protect public safety.

Key 177

A Case Study: Raised Intracranial Pressure Without a Mass Lesion?

A 30-year-old woman visits the neurology clinic complaining of worsening headaches over the past two months. She describes the headaches as diffuse and pressure-like, which worsen when lying down and improve slightly when sitting upright. She also reports occasional episodes of blurred vision, especially when moving from sitting to standing. During these episodes, her vision turns grey but returns to normal after a few seconds. Her body mass index (BMI) is 37 kg/m². She has no history of recent trauma, neck stiffness, or fever. On examination, she is found to have bilateral papilloedema, but her cranial nerve function is intact. Blood pressure is within normal limits, and a recent MRI of the brain showed no mass lesions or other abnormalities. What is the most appropriate next step in the investigation?

- A) CT venogram.
- B) Coagulation profile.

- C) Lumbar puncture.
- D) Blood glucose measurement.
- E) Visual field testing.

Answer:

The correct answer is \rightarrow **C)** Lumbar puncture.

Explanation:

- Symptoms and Examination: This patient presents with symptoms and signs suggestive of idiopathic intracranial hypertension (IIH), such as headaches, visual disturbances, and papilloedema. These symptoms, combined with the absence of any mass lesion on MRI, indicate raised intracranial pressure (ICP) without any obvious structural cause.
- MRI with contrast or MR venogram: These are the first-line imaging
 investigations to exclude a mass lesion or hydrocephalus in cases of
 suspected idiopathic intracranial hypertension (IIH). If an MRI is unavailable, a
 CT scan can be performed, though it is less sensitive.
- Lumbar puncture: Once structural abnormalities have been excluded by imaging, the next step is to perform a lumbar puncture. This measures the

opening pressure, confirming raised intracranial pressure, which is diagnostic of IIH. The cerebrospinal fluid (CSF) can also be analysed to rule out infections or other causes, such as **subarachnoid haemorrhage**.

Visual Field Testing (Option E): While important for monitoring papilloedema
and potential visual loss in IIH, visual field testing is more useful after
confirming the diagnosis and monitoring progression, rather than as the initial
step.

Idiopathic Intracranial Hypertension (IIH)

Idiopathic Intracranial Hypertension (IIH), also known as **pseudotumor cerebri**, is a condition characterised by increased **intracranial pressure** (ICP) without a detectable mass lesion or hydrocephalus. It most commonly affects **obese women of childbearing age**.

Clinical Presentation:

• **Headaches**: Typically throbbing, **pressure-like** and worsened when lying down or bending over. Patients often describe the pain as more severe in the morning.

- Visual disturbances: This includes transient visual obscurations, where vision momentarily dims or <u>blurs</u>, especially when changing positions. Papilloedema can also result in gradual visual field loss.
- **Bilateral papilloedema**: A hallmark sign of raised ICP, often identified during fundoscopic examination.
- Other symptoms: Patients may experience nausea, vomiting, or even tinnitus.

Investigations:

- MRI with contrast or MR venogram: These are the first-line imaging investigations to exclude a mass lesion or hydrocephalus in cases of suspected idiopathic intracranial hypertension (IIH). If an MRI is unavailable, a
 CT scan can be performed, though it is less sensitive.
- Lumbar puncture: Once structural abnormalities have been excluded by imaging, the next step is to perform a lumbar puncture. This measures the opening pressure, confirming raised intracranial pressure, which is diagnostic of IIH. The cerebrospinal fluid (CSF) can also be analysed to rule out infections or other causes, such as subarachnoid haemorrhage.
- Visual field testing: Although not the primary diagnostic step, visual field testing can be done to assess the extent of visual impairment due to

papilloedema and monitor the progression of **visual field loss**. This is especially important for follow-up after the diagnosis.

These investigations help establish the diagnosis of IIH and rule out other conditions that could cause similar symptoms, such as **venous sinus thrombosis** or **mass lesions**.

■ *Management:*

- Weight loss: One of the most effective treatments in patients with obesity.
- Medications: <u>Acetazolamide</u> is commonly used to reduce CSF production and lower ICP. <u>Topiramate and Prednisolone</u> may also be beneficial.
- Procedures: Repeated lumbar punctures or optic nerve sheath fenestration
 may be performed to alleviate symptoms and prevent visual loss in severe
 cases.

Idiopathic intracranial hypertension requires close monitoring, especially of visual function, as untreated cases may lead to permanent vision loss.